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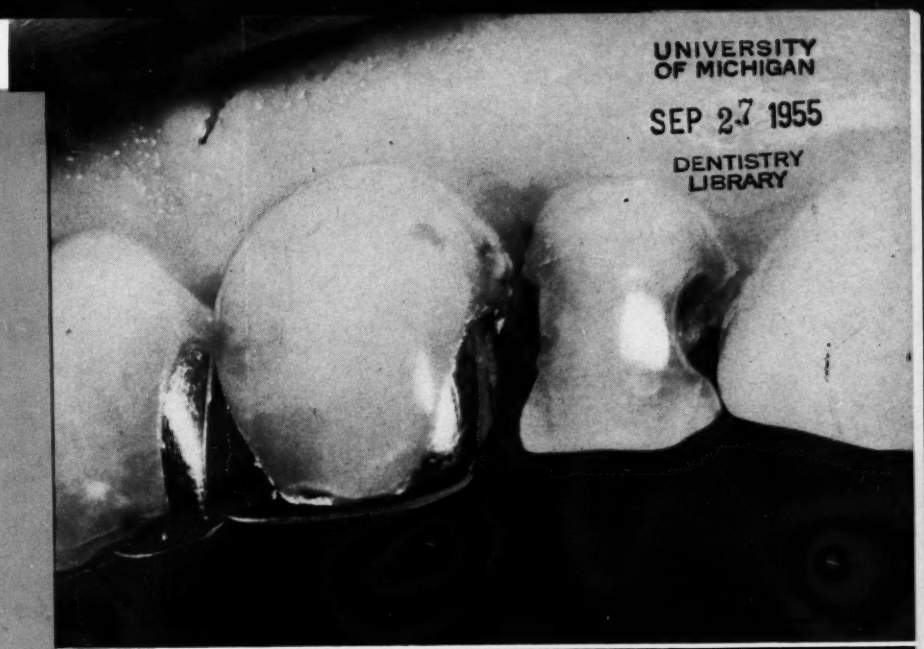

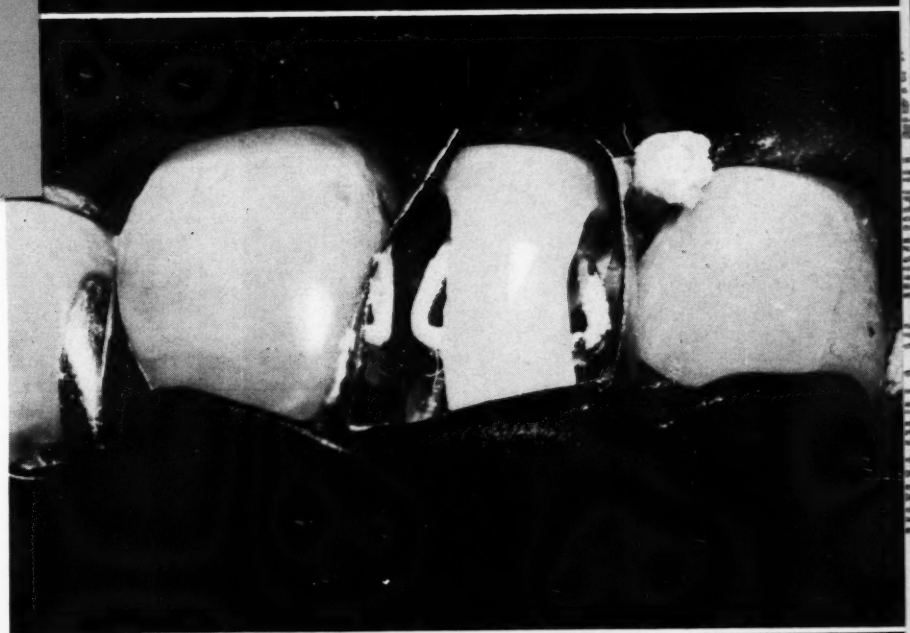

September 1955

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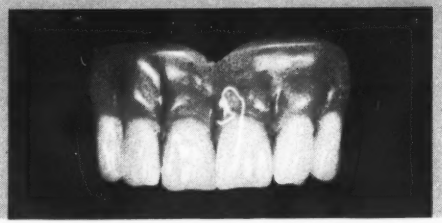

This descriptive symbol simplifies tooth selection... M42/C

M—MEDIUM LENGTH
42—WIDTH IN MILLIMETERS OF SIX ANTERIORS CARDED FLAT
C—DOMINANTLY "C" CURVED LABIAL CHARACTERISTICS

SCHEMATIC DRAWING OF
FIVE-PHASE ANTERIOR CO-ORDINATE SIZE MOLD SYSTEM

WIDTH OF 6s CARDED FLAT	36 MM	39 MM	40 MM	42 MM	43 MM	45 MM	46 MM	48 MM	51 MM
LONG	L36/C	L39/C L39/F		L42/C L42/F		L45/C L45/F		L48/C L48/F	L51/C
MEDIUM	M36/C	M39/C M39/F	M40/C M40/F	M42/C M42/F	M43/C M43/F	M45/C M45/F	M46/C M46/F	M48/C M48/F	M51/C
SHORT		S39/C S39/F	S40/C S40/F	S42/C S42/F	S43/C S43/F	S45/C S45/F	S46/C S46/F	S48/C S48/F	

FIVE FEATURES THAT ASSURE ACHIEVEMENT OF "LIVING" BEAUTY

1. You can select Five-Phase Anteriors with dominantly flat or dominantly curved labial characteristics.
2. You can transpose centrals, laterals and cuspids from different sets, when required—a feature that is unique to Five-Phase Anteriors.
3. You can use more than one tooth color in the same denture.
4. You can achieve maximum flexibility in tooth selection and arrangement.
5. You can select Five-Phase Anteriors in your choice of either Veri-chrome Porcelain or Verident Plastic.

Tooth selection can be simple and logical. Actual length, width and labial character required for each case *automatically* determine the Five-Phase Anterior mold to specify.

Five-Phase Anteriors are arranged in the Co-ordinate Size Mold System shown on this schematic chart. As you can see, standard sets are available in a choice of dominantly flat (F) or dominantly curved (C) labial character . . . in a full range of widths from 36 mm to 51 mm (the measurement of the six anteriors carded flat) . . . in long, medium and short molds.

In addition, the system is extremely flexible. The proximals of all Five-Phase Anteriors are co-acting. Choose from carded sets with the individual labial characteristics required or transpose teeth from different sets to create personalized dentures. The logical, orderly arrangement of the Five-Phase Coordinate Size Mold System makes this so easy to do.

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Vol. 61, No. 9

Dental Digest

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RETENTION SIMPLIFIED

Employing Prefabricated Tapered Gold Pins—Part One

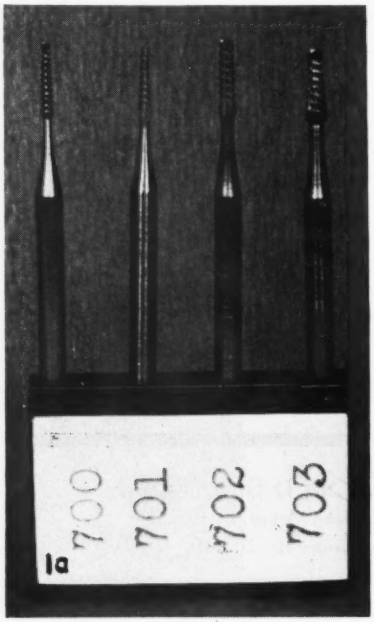
M. M. CHECHIK, D.D.S., Vancouver

DIGEST

The basic principle in this technique is the use of prefabricated gold pins which will positively fit a hole prepared by any standard tapered bur. Tapered pins are employed so that any discrepancy or looseness of fit can be compensated by grinding part of the smaller end of the taper thereby permitting the pin to wedge further against the walls of the preparation.

The use of pins for restoring broken down teeth is by no means new. These prefabricated pins, however, simplify and improve the technique.

In this installment, the first of two parts, the procedure applied in three case histories is illustrated.



1A. ***The various burs used are shown.***

Preparation of Gold Pins

The original technique for forming prefabricated pins of sizes 700, 701, 702, and 703 was the following:

1. Burs of the numbers mentioned made by the same manufacturer were obtained so that all the burs had the same degree of taper (Fig. 1a). Thus any tapered hole from the smallest diameter of the 700 to the largest diameter of the 703 could be fitted by one of these prefabricated pins.

2. A machinist was employed to form dies by drilling a small brass rod with the above burs.

3. From these dies wax patterns were secured and cast in gold.

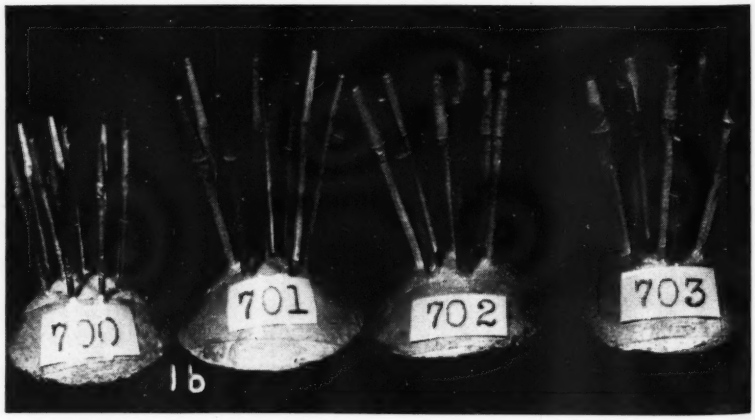
4. This procedure provided pins of the exact dimensions required. Figure 1a shows the various burs used. Figure 1b shows castings of various pins.

The tedious procedure described is no longer necessary since these pins are now manufactured and available through the dental supply houses.*

Use of the Pins

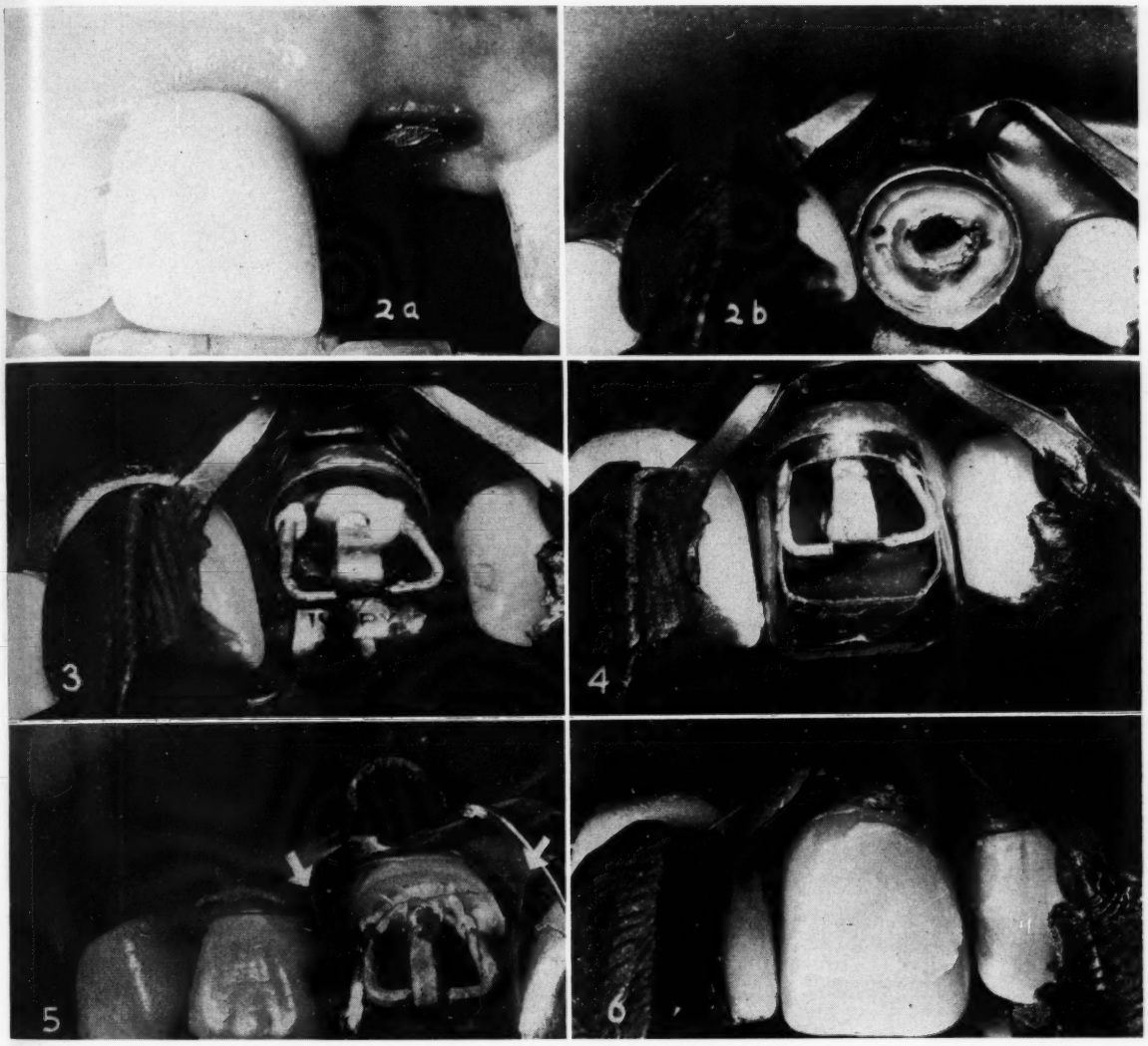
The chief use is to secure retention with autoset resins where there is insufficient tooth structure to retain restorations, as in the following:

(a) To restore fractured anteriors



1B. ***The castings of various pins.***

*Wilkinson Company, Santa Monica, California.



2A. *The patient in Case One presented with a loose fitting dowel crown which was removed.*

2B. *This tooth had received root canal therapy. It was therefore possible to prepare a No. 703 bur hole as well as two auxiliary 700 bur holes. In addition, a groove was prepared just inside the dentocemental line to increase retention.*

3. *A No. 703 prefabricated pin was selected and bent lingually to conform to the lingual surface of the restoration. The two auxiliary 700 pins were bent over at right angles toward the central pin. These were then cemented in place.*

4. *The crown form was held in position and blocked with compound.*

5. *Where contact points are required the crown forms are disced mesially and distally as thin as possible without cutting through. Two wedges are then firmly inserted proximally to minimize the overflow at the gingival and to compensate for the remaining thickness of the crown form. It is similarly blocked with compound.*

6. *The patient is placed in a position so that the labial surface of the tooth is horizontal. The autoset resin is mixed and poured into the crown form through the open window and allowed to set.*

without pulpal involvement

(b) To restore incisal angles or Class IV cavities

(c) To restore cusps on cuspids and bicuspids

(d) To restore part of, or all of the crown of devitalized anteriors and bicuspids

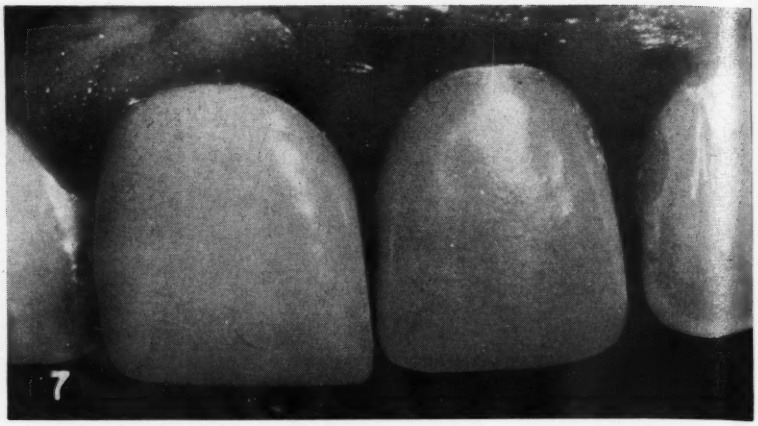
Case One—Procedure to Restore a Full Crown on an Upper Devitalized Central

The patient presented with a loose fitting dowel crown, which was removed (Fig. 2a). The following steps were then taken:

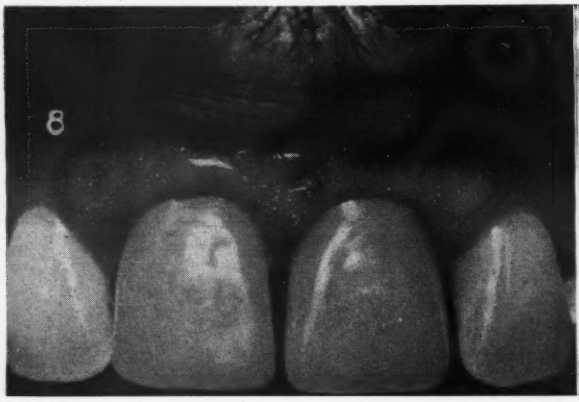
1. A copper collar was fitted over the gingival shoulder of the remaining portion of the tooth to facilitate the placement of the rubber dam and clamp. This procedure is not always necessary but was used in this case because the gingival shoulder on the lingual was so deep subgingivally.

2. This tooth had received root canal therapy. It was therefore possible to prepare a No. 703 bur hole as well as two auxiliary 700 bur holes. In addition, a groove was prepared just inside the dentocemental line to increase retention (Fig. 2b).

3. A No. 703 prefabricated pin was selected and bent lingually to conform to the lingual surface of the restoration. The two auxiliary 700 pins were bent over at right angles toward the central pin. These were then cemented in place (Fig. 3). If one wishes to eliminate the plunger action of cementing the pins, one can cut a small groove with a small wheel bur, or a small Jo Dandy disc, or otherwise with a stone, grind down a small portion of one side to allow an escape for the cement. This will allow the pin to be readily seated in position.



7. *The resin is trimmed to form and polished.*



8 *and* **9.** *The same case (Case One) at a later date with the approximating central restored, employing the same technique. Note outline of pins.*

4. It may be necessary to paint the gold pins with an opacifier. A crown form for use with autoset resins is selected for the proper form and size and is trimmed to fit over the shoulder of the prepared tooth. In this particular case we were not concerned with the contact points. The crown form was therefore held in position and blocked with compound (Fig. 4).

(Where contact points are required the crown forms are disced mesially and distally as thin as possible without cutting through. Two wedges are then firmly inserted proximally to minimize the overflow at the gingival and to compensate for the remaining thickness of the crown form. It is similarly blocked with compound (Fig. 5).)

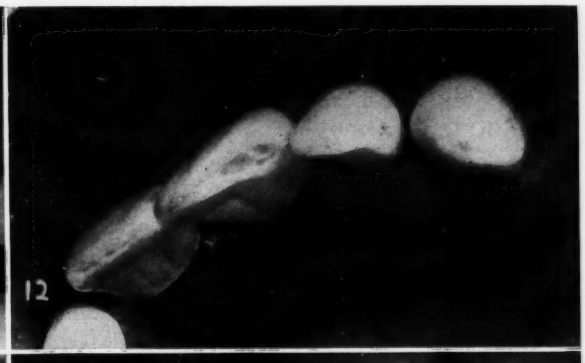
5. Into the labial of the crown form which is blocked into position, a window is cut to provide access. Sufficient bulk of the crown, however, is left to retain its form. This can be done by starting with a sharp gold knife and finishing with an extremely small pair of cuticle scissors (Fig. 4).

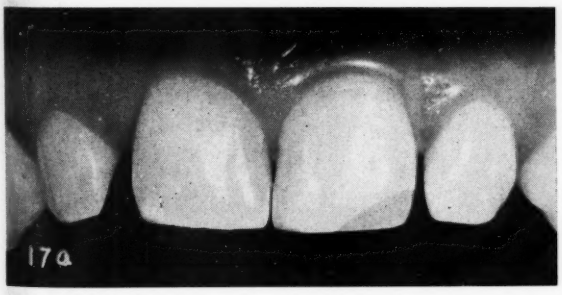
6. The patient is placed in a position so the labial surface of the tooth is horizontal. The autoset resin (Sevriton®) is mixed and poured into the crown form through the open window and allowed to set (Fig. 6). This technique applies to the type of autoset resin which can either be flowed in or brushed in.

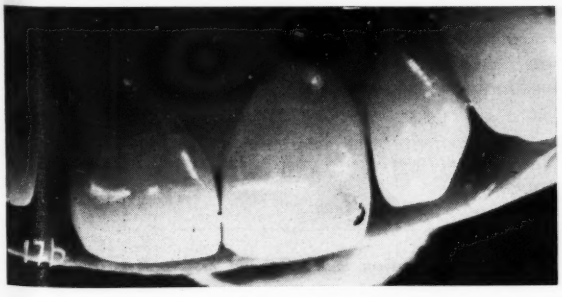
10. *An x-ray view showing the pins in position on the completed case.*

7. The resin is trimmed to form and polished (Fig. 7).

8. The same case at a later date with the approximating central re-







11. *The patient in Case Two presented with a distal incisal angle fractured off on the upper left central.*

12. *Under the rubber dam the irregularities of the fracture line were eliminated and an incisal and axial pinhole were prepared with a No. 700 bur.*

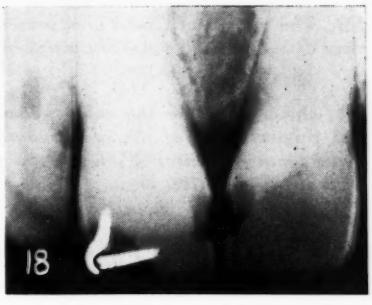
13. *and* **14.** *Suitable pins were selected, contoured, and cemented into position.*

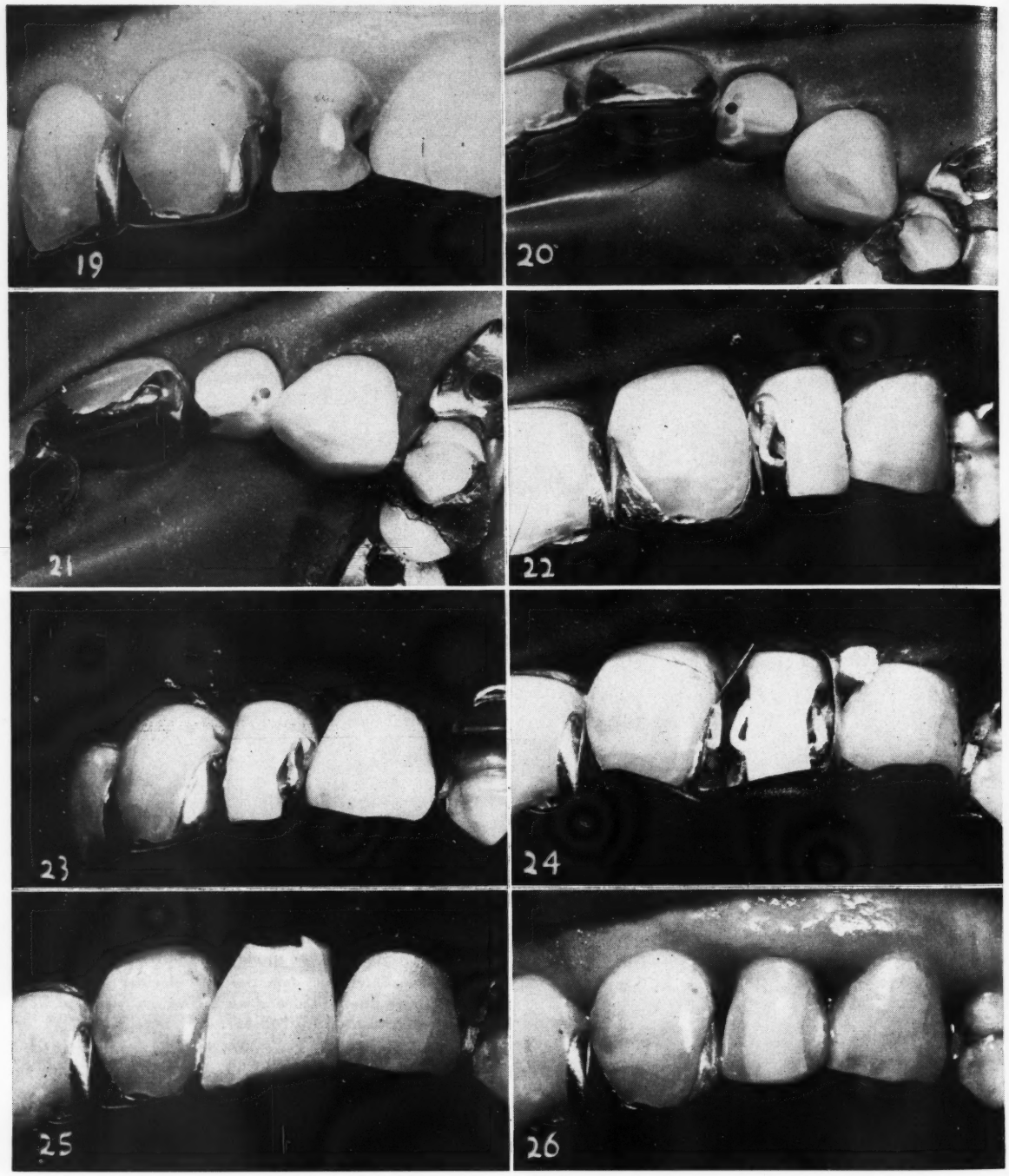
15. *A gold knife may be employed to prepare a window in the crown form of slightly smaller dimension than the area involved.*

16. *The autoset resin was mixed and flowed through the window, filling the preparation.*

17A. *and* **17B.** *The resin was trimmed and polished.*

18. *An x-ray view showing pins in the completed case.*





19. *The patient in Case Three presented with an involvement of both incisal angles of the upper lateral.*

20. *and* **21.** *Under the rubber dam, the labial, lingual, and gingival walls of the preparation were established in sound tooth structure. A pinhole was prepared on the gingival seat with a 700 bur.*

22. *and* **23.** *Suitable pins were selected, contoured, and cemented in position.*

24. *To retain the restorative material the matrix metal was cut at the mesial and distal incisal and the cut portion was bent over the incisal edge. It was then blocked into position with compound.*

25. *The autoset resin was mixed and flowed into the preparation.*

26. *The restoration was trimmed and polished.*

stored, employing the same technique. Note outline of pins (Figs. 8 and 9).

9. An x-ray view showing the pins in position on the completed case (Fig. 10).

Variations in Technique—When it is desired to use those autoset resins which do not lend themselves to the application described, the technique may be varied as follows:

1. After fitting the crown form, it is held in position, lubricated, and compound is applied lingually and just over the incisal edge.

2. It is chilled and pulled away from the crown form.

3. Try in position to make sure the key locks the crown form into the previously held position. This compound key is set aside.

4. Small escape holes can be made into the mesial and distal incisal angles of the crown form. The resin is mixed and packed into the crown form which is then carried over the prepared tooth. The compound key is locked into place to locate the proper position of the crown form.

Case Two—Procedure to Restore Fractured Anteriors Without Pulpal Involvement

The patient in this case presented with a distal incisal angle fractured off on the upper left central (Fig. 11). The following steps were taken:

1. Under the rubber dam the irregularities of the fracture line were eliminated and an incisal and axial pinhole were prepared with a No. 700 bur (Fig. 12)

27. ***An x-ray picture showing the completed case. Note the position of the pins.***

2. Suitable pins were selected, contoured, and cemented into position (Figs. 13 and 14).

3. The opacifier was applied to the gold pins and a suitable crown form was placed in position over the tooth and blocked with compound. A gold knife may be employed to prepare a window in the crown form of slightly smaller dimension than the area involved (Fig. 15).

4. The autoset resin was mixed and flowed through the window, filling the preparation (Fig. 16).

5. The resin was trimmed and polished (Figs. 17a and 17b).

6. Figure 18 is an x-ray view showing pins in the completed case.

Case Three—Procedure to Restore a Class IV Cavity

In this case the patient presented with an involvement of both incisal angles of the upper lateral as shown in Figure 19. The following steps were included in the restorative procedure:

1. Under the rubber dam, the labial, lingual, and gingival walls of the preparation were established in sound tooth structure. A pinhole was prepared on the gingival seat with a 700 bur (Figs. 20 and 21).

2. Suitable pins were selected, contoured, and cemented into position (Figs. 22 and 23).

3. The opacifier was applied. A portion of metal matrix band material was contoured by burnishing with a beaver tail burnisher. This band material was placed in position and wedged firmly.

4. To retain the restorative material the metal was cut at the mesial and distal incisal and the cut portion was bent over the incisal edge. It was then blocked into position with compound (Fig. 24).

5. The autoset resin was mixed and flowed into the preparation (Fig. 25)

6. The restoration was trimmed to form and polished (Fig. 26).

7. An x-ray picture showing the completed case. Note the position of the pins (Fig. 27).

(*End of Part One*)

923 *Denman Street*

Training of Dental Hygienists

DURING THE last decade people have become increasingly aware of the need for continuous and better dental care. This awareness is already paying health dividends but it also has resulted in heavy demands upon the time and energy of the practicing dentist. Obviously, these demands have placed a premium upon the services of dental hygienists, dental assistants and dental laboratory technicians, for such auxiliary personnel relieve the dentist of many routine tasks and allow him to extend his services to more patients.

From *Annual Report*, W. K. Kellogg Foundation, 1953-1954, page 57.

REACTIONS TO LOCAL ANESTHESIA:

Types, Manifestations, and Treatment

RONALD F. STEGELSKE, D.D.S., Erie, Pennsylvania
and WILLIAM R. GRILL, D.D.S., Baltimore, Maryland

DIGEST

Considering the number of local anesthetics used daily without severe reactions, it would appear that they are relatively safe. On rare occasions, however, serious side effects may occur. Although much time is spent teaching the pharmacology and technique of local anesthesia, it is possible that not enough emphasis is placed on the emergency treatment necessary if an unsatisfactory reaction should occur. This article reviews the literature on this subject and discusses the various types of reactions, their manifestations, and their prophylaxis and treatment.

Classification of Reactions

In forming a classification of the types of reactions which may occur after the injection of local anesthetics several other arrangements have been coordinated.[1,2,3] The classification is as follows:

1. Circulatory
   - (a) Mild (psychomotor)
   - (b) Complete collapse
   - (c) Epinephrine
2. Convulsive
   - (a) Stimulation of the central nervous system
   - (b) Severe asphyxiation
3. Respiratory
   - (a) Depression of the central nervous system
4. Allergic
   - (a) Contact dermatitis
   - (b) Serum sickness pattern reaction
   - (c) Accelerated serum sickness
   - (d) Atopic reaction

Circulatory Reactions—These are the reactions which predominantly affect the blood vascular system and manifest themselves in various other ways.

Psychomotor or Mild Circulatory Reaction: This is perhaps the most common of all. Most of these reactions are not due to the drug injected but rather to the action of the vasovagal reflex[3] which originates in the higher brain centers as a result of apprehension or fear and (1) produces peripheral vasodilation, (2) lowers peripheral resistance and blood pressure, and (3) may produce syncope. The patient feels dizzy or weak, is pale, has a rapid pulse and may lose consciousness. The patient who faints at the sight of a needle would be an example of this reaction.

Cardiac Failure: Circulatory collapse can occur from an accidental injection into the blood stream or a vascular area. In this instance the heart is affected before the brain and the drug acts directly on the heart muscle resulting in cardiac failure. (1) The patient may lose consciousness without warning, (2) the pulse cannot be detected, and (3) there is no blood pressure. Breathing usually continues although it may be rapid and gasping. There is no cyanosis. The drop in circulating blood volume results in a hemoconcentration in the peripheral circulation much more severe than in syncope and shock usually ensues. Cardiac failure with collapse can be a dangerous side effect for the symptoms occur quickly and death can result.

Epinephrine: Although epinephrine is not a local anesthetic, it is a part of most solutions and is therefore included in the classification. The following are some of the features in epinephrine reaction:

1. Epinephrine can stimulate the myocardium and cause an excititory effect with palpitation, tachycardia, nervousness, and possibly convulsions. These symptoms are usually transient and not serious except in coronary artery disease where coronary heart failure may result.
2. The drug is also contraindicated in hyperthyroid patients and vasabile subjects. Epinephrine and local anesthetic reactions can be differentiated.[4]
3. Epinephrine reaction develops more rapidly, is not as persistent, and recedes faster. There is some conflict on this issue however.

Convulsive Reactions—When an anesthetic agent reaches the brain in sufficient quantities to stimulate the motor area of the cerebral cortex, convulsions usually result. This reaction occurs most often from an accidental intravenous injection.

Stimulation of Central Nervous System: Nervousness, anxiety, fear, muscular tension, trembling, tics, and hallucinations with or apart from the convulsions may be produced. The fear reaction often results in a sympathetic stimulation and an exaggeration of the original symptoms. Convulsions usually carry hypoxia and tachycardia with them although res-

[1]Sadove, M. S.; Wyant, S. M.; Gittelson, L. A.; and Kreschner, H. E.: Classifications and Management of Reactions to Local Anesthetic Agents, JAMA **148**:17-22, (Jan. 5) 1952.
[2]Criep, L. H.: Reactions to Procaine Hydrochloride, JADA **47**:149-153 (August) 1953.
[3]Nevin, M. I.: Diagnosis and Treatment of Anesthetic Reactions, Modern Dentistry **21**:7-11 (July) 1954.
[4]Shotwell, J. E.: Toxic Effects Following the use of Local Anesthetic Agents, Oral Surg; Oral Med; Oral Path. **1**:730-735 (Aug.) 1948.

piration and circulation are essentially not interfered with.

Severe Asphyxiation Initiated by Medullary Depression: Stimulation of the spinal cord and other centers may bring about a rise in blood pressure and convulsions. This situation can easily be misinterpreted as the beginning of a stimulatory action.[5] Therefore, although convulsions are almost always the result of a central nervous system stimulation, they can occur after depression.

Adequate Treatment Important: Convulsions are often encountered but they are usually of short duration and if treatment is immediate are not usually dangerous.

Respiratory Reactions: Depression of the central nervous system, particularly the vital medullary centers result in reactions affecting respiration. This effect is almost always severe and usually occurs after inhalation or intravenous anesthesia, almost never after local anesthesia injections.

Signs: The following signs are noted: (1) A sudden cessation of spontaneous breathing, (2) unconsciousness, and (3) a rapid development of cyanosis with a normal or slightly fast pulse. Symptoms appear quickly and occur with a small or a large dose. Depression may follow or accompany stimulation for some degree of stimulation, however slight, invariably precedes or accompanies such a depression. A respiratory reaction is a dangerous one and death can result from anoxia.

Allergic Reactions in Dental Anesthesia: These are extremely rare and are therefore not as important as the other effects. They do occur occasionally, however, and are therefore included in the list.

Definition: Allergy has been defined as an altered reactivity of the tissues caused by the formation and deposit of specific antibodies. In a case where there is no history of previous local anesthesia, sensitization may have been caused by related drugs.

Contact Dermatitis: This type of reaction is more often seen in the dentist than in the patient. It is a localized contact, eczematous allergic dermatitis, and can be a detriment to the dentist's personal appearance as long as it remains.

Serum Sickness Pattern Reaction: An effect of this sort rarely occurs from local anesthesia and when it does it is after the administration of the drug for the first time. The symptoms, appearing in from one to twelve days are the following: fever, hives, joint disturbances, and general malaise.

Accelerated Serum Reaction: This reaction is much more serious than the previous one and may be fatal. Unlike the serum sickness pattern reaction, this type follows the administration of an agent for the second or a subsequent time. It is characterized by sudden development of urticaria, pruritis, dyspnea, tachycardia, sweating, cyanosis, fall in blood pressure, and subsequent shock and circulatory collapse.

Atopic Reaction: This reaction has a sudden onset with nasal and asthmatic manifestations, generalized angioedema, and urticaria. It is rare but serious as death can result due to asphyxia from bronchial obstruction.

Comment: The allergies in general are rare after local anesthesia and when they occur they are characterized by skin eruptions. Although they are infrequent it is important to recognize them and to be prepared in case an emergency should occur.

Factors in Treatment

The most efficient measure in counteracting the toxic effects of a local anesthetic agent is the proper prophylactic treatment.[6] In planning the proper treatment the factors which influence the toxicities of the local agents must be considered:[7]

1. *Susceptibility of the Patient*—The patient should be questioned regarding a previous experience with local anesthesia and any existing allergies or disease that might influence the selection of a local anesthetic agent.

2. *Rate of Injection*—The rate of injection can be controlled by the operator. If the injection is given rapidly the anesthetic solution will be forced more readily into the blood vessels and lymphatics, thereby achieving the equivalent of an intravenous injection. By slowly injecting the solution the drug will have an opportunity to diffuse through the tissues and become localized through the development of vasoconstriction.

3. *Vascularity of the Part Injected*—The vascularity of the site of injection greatly influences the rate of absorption of the anesthetic drug into the blood stream and the blood level of the drug is presumably an important factor in the causation of toxic reactions.

4. *Concentration of the Solution*—The toxicity of a local anesthetic drug increases in geometrical progression with the increase in concentration of the anesthetic solution.

5. *Total Weight of the Drug Injected*—The dosage by weight of each local anesthetic drug varies with the physical status, age, state of nutrition of the subject. For a normal healthy person there is an average dosage of each drug by weight; any increase in this average dose is likely to result in unsatisfactory reactions.

Toxic Reactions Limited—Of the factors discussed, the susceptibility of the patient or the vascularity of the site of injection cannot be controlled. By using the smallest amount of an anesthetic drug of the lowest concentration and injecting slowly into the tissue, many toxic reactions, however, can be altered or eliminated entirely. If the patient relates a history of a toxic reaction to an anesthetic drug, other such experiences may be avoided by using an anesthetic drug having a different chemical structure.[5]

Fears and Apprehensions Allayed—In many dental patients fear and apprehension prior to treatment may produce mild circulatory symptoms following the injection of a local anesthetic. This type of reaction is due to the vasovagal reflex which has been described earlier in this article. By a proper psychologic approach to the patient before the injection, fear or

[5]Keaveny, J.: Clinical Aspects of Local Anesthetics, JADA **29**:755-762 (May) 1942.

[6]Hickey, M. J.: Local Anesthesia in Oral Surgery, JADA **33**:1532-1540 (Dec.) 1946.

[7]Lum, R.: Antidotes for Procaine Toxicity, "Contact Point" **20**:89-92 (Sept.) 1942.

apprehension can be allayed and to a great extent the frequency of the mild circulatory reactions can be controlled.

Patient Confidence Gained—Deliberation in the injection procedure and a sufficient waiting period for satisfactory anesthesia before beginning operative procedure, will win the confidence of the patient and abolish his fear of pain which could precipitate a mild type of circulatory disturbance.

Premedication May be Required—Despite competent psychologic approach, some patients may require premedication to overcome fear of the ensuing dental procedure. In these cases the use of a drug that will promote relaxation and mental tranquility in a patient will reduce the frequency of a toxic reaction and also improve the operating conditions for the dentist.

Commonly Used Drugs—The barbiturate group of sedative compounds are the most commonly employed for the premedication of apprehensive patients. The most frequently used in this group are the following:

Pentobarbital (1½ - 3 grains)
Phenobarbital (1½ - 3 grains)
Secobarbital (1 - 3 grains)
Amobarbital (1½ - 4 grains)
Hexobarbital (4 - 6 grains)

Other drugs which may be used are meperidine (1 - 2½ grains) and carbromal (5 - 15 grains). For the child patient the drug dosage varies with the weight of the child.

Procedure in Local Anesthetic Administration

Despite the care taken in preparing a patient for local anesthesia, all the possibilities of toxic reactions cannot be eliminated. For this reason the dentist must constantly be prepared to treat these reactions as quickly as they are recognized. By careful planning serious consequences may be prevented.

Treatment in Mild Circulatory Reaction—When a mild circulatory or an epinephrine type of toxic reaction is recognized by the symptoms of a patient after an injection (1) the patient should be placed in a supine position, (2) tight clothing loosened, and (3) a mild stimulant such as aromatic spirits of ammonia or any nitrite given to the patient to inhale. Aromatic spirits of ammonia may also be given orally in water. This is usually sufficient to prevent the patient from losing consciousness. The same treatment is used in syncope and the patient should respond quickly. After recovery is complete the operative procedure may be resumed.

Complete Circulatory Collapse—Place the patient in the supine position and administer oxygen. Epinephrine injection subcutaneously will stimulate the heart. This injection may be repeated in 10 to 15 minutes if necessary. In complete cardiac failure the heart beat may be started by striking the chest sharply over the heart.[3] The final measure and most drastic in cases of cardiac failure is to massage the heart directly through an incision into the thoracic cavity.

Symptoms of Convulsions—If the patient shows the symptoms of convulsive movements after an injection of a local anesthetic drug, a depressant drug such as sodium amytal (5 per cent) or sodium pentobarbital may be injected intravenously until the convulsions cease. The patient may also be supported by the administration of oxygen.

Artificial Respiration—When there is a sudden cessation of breathing, oxygen is administered immediately. If oxygen is not available, artificial respiration should be started without delay. As an adjunct a respiratory stimulant such as metrazol (10 per cent) can be given subcutaneously.

Cases of Toxic Reaction—There is no possibility of error when pure oxygen is administered to the patient and an adequate airway is ensured.

Treatment in Types of Allergic Reactions—Most instances of allergic reaction do not require emergency treatment. It is well to be prepared however.

Contact Dermatitis: This condition can be avoided rather than treated. The patch test is believed by some to be an aid in determining an allergy. The majority of investigators, however, feel that it is of little value when dealing with a local anesthetic agent. Antihistamines are usually employed in the treatment of contact dermatitis.

Serum Sickness: This delayed reaction is usually treated with ACTH, epinephrine, and antihistaminics. An allergic reaction demanding immediate treatment is accelerated serum sickness which is most successfully treated by intramuscular injections of antihistamines or epinephrine and the administration of oxygen and a sedative.

Atopic Response: This condition also requires immediate care and is most successfully treated by subcutaneous injections of epinephrine and the administration of oxygen.

Summary

The classification, prophylaxis, and treatment of the toxic reactions of local anesthetic drugs employed daily in the practice of dentistry have been discussed.

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Authors' Note: The views stated in this paper are those of the authors and do not reflect the views of the United States Air Force.

[8]Tainter, M. L.: Prevention and Treatment of Local Anesthetic Reactions, J. California Dental A. and Nevada D. Soc. **28**:6-10, (Nov.-Dec.) 1952.
[9]Criep, L. H.: Allergy to Procaine Hydrochloride with Three Fatalities, JAMA **151**: 1185-1187, (April) 1953.
[10]Dick, S. B.: Clinical Toxicities of Epinephrine Anesthesia, Oral Surg.; Oral Med.; Oral Path. **6**:724-728 (June) 1953.
[11]Dobbs, E. C.: Local Anesthetic Drugs, J. Baltimore Coll. D. Surg. **9**:45-49 (Nov.) 1946.
[12]Tainter, M. L.: Causes and Treatment of Systemic Reactions in Local Anesthesia, JADA **20**:152-155 (Jan.) 1953.
[13]Walters, J. D.: Toxicity of Local Anesthetics, Navy Med. Bull. **48**:871-877 (Nov.-Dec.) 1948.

OCCLUSAL MOVEMENTS *and* OBSTRUCTIONS:

the Bennett Movement – Part Two

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DIGEST

In this installment which is the second of two articles on occlusal movements and obstructions, the author discusses the Bennett movement demonstrating that the movement is a component of vertical instead of lateral motions.

The Bennett Movement

The Bennett Movement is still a moot subject in articulation. Granger[4] describes it as ". . . the medial gliding of the condyle on the working side . . ." He credits his readers with realizing that this would entail a "medial gliding" of the entire jaw since one condyle moving medially would force the other to move radially. It must also be assumed that he uses medial here in its true sense; meaning toward the midline or center and not in the loose sense that indicates motion in a forward direction.

Concise Definition — Lucia[5] describes the Bennett movement more concisely as ". . . the side-to-side movement of the mandible." Granger and Lucia are in agreement that the Bennett occurs in lateral motion only. Both express forcefully their assumptions of the *functional* importance of the Bennett movement.

Opinions of Bennett Movement Vary—1. Granger's[2] emphatic opinion of the role of the Bennett movement in function has already been quoted.

2. Lucia[5] states, "The Bennett movement is the jaw movement that the patient uses to triturate food."

3. McCollum agrees that the Bennett is a component of lateral motion but his views on its direction of movement and its functional importance are at considerable variance and appear much more logical. McCollum[6] states, "The Bennett path . . . may not go directly laterally, but may slope upward, downward, backward or forward," and adds, ". . . these directions may exist in combination . . ."

4. McCollum's experiments also convinced him[7] that ". . . some jaws do not have any Bennett movement at all and many others on one side only."

5. McCollum also sees the Bennett movement, not as the most important component in *function* but as an important deviation from an otherwise normal jaw movement that must be recognized and served by adapting cuspal surfaces and inclines. McCollum[8] states, "The shape of the Bennett path has as much as, and probably more, influence upon the articulating surfaces of the teeth than any other component of jaw motions."

Problem of One Bennett or None—If the Bennett movement is the "power movement" and "the most important in the mandibular synergy" and "the jaw movement [used] to triturate food," what of those patients who, according to McCollum, have a Bennett movement on one side only or none at all? It would appear that these persons are most unfortunate. The unilateral Bennett chewer would be able to masticate efficiently on one side only while the unfortunate patient with no Bennett at all must, perforce, resort to preground foods and liquids. He would lack entirely "the power movement" needed "to triturate food."

Authorities Agree on Cause for Movement—Both McCollum[9] and Lucia[10] give essentially the same cause for the Bennett movement. The former states: "If the inner curbing of the temporal fossa is turned inward more than a simple rotation of the opposite condyle can accommodate, the rotating condyle must move outward." Lucia states: "The glenoid fossa . . . medially . . . is bounded by a bone structure called the inner curbing of the fossa. This inner curbing has a varying curvature and inclination. It is this variation in the formation of the curbing that produces the variation in the Bennett path."

Second Theory for Cause—Sicher[11] has an entirely different theory for the cause of the Bennett movement. He too is satisfied, apparently, that it is a component of laterals only. Concerning its cause he states emphatically; "It is clear . . . that this time lag between the contraction of the [nonworking] external pterygoid muscle and the [working] temporal muscle is responsible for the Bennett movement."

According to Sicher there is a definite and vital reason for the Bennett quite different from the reasons given by the others;[12] ". . . the contraction

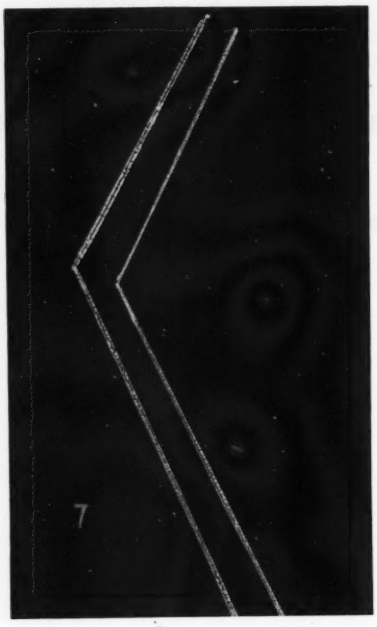
[4]Granger, Ernest R.: Centric Relation, J. Pros. Dent. **2**:169 (March) 1952.
[5]Lucia, Victor O.: The Fundamentals of Oral Physiology and their Practical Application in the Securing and Reproduction of Records to be Used in Restorative Dentistry, J. Pros. Dent. **3**:220 (March) 1953.
[6]McCollum, Beverly B.: Fundamentals Involved in Prescribing Restorative Dental Remedies. Reprint from June 1939 to February 1940 D. Items Interest, p. 48.
[7]Ibid. p 28.
[8]Ibid., p. 45.
[9]McCollum, Beverly B.: Oral Diagnosis, JADA **30**:1224 (Aug.) 1943.
[10]Lucia, Victor O.: The Fundamentals of Oral Physiology and their Practical Application in the Securing and Reproduction of Records to be Used in Restorative Dentistry, J. Pros. Dent. **3**:221 (March) 1953.
[11]Sicher, Harry: Positions and Movements of the Mandible, JADA **48**:625 (June) 1954.
[12]Ibid.

of the retracting fibres of the [working] temporal muscles occurs after an initial swing of the entire mandible laterally and anteriorly to avoid strain in the capsule that would be caused by a rotation of the resting condyle in situ."

Contradiction Presented—Sicher's theory presents a complete contradiction to the results portrayed by McCollum's experiments. If Sicher is right and it is essential that there be a Bennett movement "to avoid strain in the capsule" then the Bennett would logically exercise this vital protection *in both joints and in every jaw*. This would indicate either that McCollum's experiments were poorly executed or his results were incorrectly interpreted.

Lateral Movements Demonstrated—Conclusions should not be formed hastily, however: (1) McCollum[13] actually completed his experiments and published them fully, and (2) for over twenty-five years members of his Gnathological Society have been and still are writing lateral jaw movements daily and are consistently demonstrating that whatever they may consider to be a Bennett movement in lateral is frequently unilateral. It is justified, therefore, to ask whether Sicher has done any experimenting to substantiate his assertions or whether he is merely exercising professorial prerogatives. If he has made any tests he does not mention them.

Questions Unanswered—There are points not made clear in Sicher's explanation: (1) Why should a "resting" joint "in situ" and functioning in a perfectly natural and normal manner be subject to such strain that it requires outside aid for relief? and (2) just how would a Bennett movement act to mitigate this assumed strain? It would be the nonworking external pterygoid that would activate the working (Sicher calls it the "resting") condyle and this external pterygoid operates, as he admits, protrusively and radially with respect to the working condyle. Consequently, there could be only one Bennett path for the working condyle; namely, obliquely outward and forward. This would apply to every jaw.



7. ***Bennett movement resulting from 1½ millimeters of opening.*** **(*See text.*)**

One-track Bennett Movement Impossible Mechanically—A universally one-track Bennett movement not only seems illogical physiologically but this particular one is definitely *impossible mechanically*. The nonworking external pterygoid would operate here as a force similar to any peripheral force that spins the rim of a wheel while the retracting fibres of the working temporal would have the mechanical advantage of being the central holding force that maintains the position of the axle.

Illustration—A four-year-old child running with a pinwheel on the end of a stick illustrates how little central holding power is required to maintain axle position against peripheral torque and therefore how futile would be any attempt of the nonworking external pterygoid to force or maintain a jaw position against the holding power of the retracting fibres of the working temporal.

Theory of Timing in Muscle Action—Sicher describes this muscular "time lag" and the "initial swing" of the jaw as illustrating "... another principle in muscle action, that of split second timing." He goes on to show how one muscle will contract or relax just a fraction of a second before or after relaxation or contraction of its opponent; something that is necessary in muscle coordination.

Problem is Presented—Does Sicher's explanation suggest that the condyle is snapped out of its dangerous position "in situ" at the instant lateral motion starts only to be snapped back a "split second later" to finish the major part of the radial lateral stroke? This hardly seems logical yet the only alternative would appear to be that the condyle maintain its outward and forward position throughout the stroke, something already shown to be impossible.

Protecting Against Strain in Mastication—The natural question of protection against strain on the returning or medial lateral stroke arises. Here, if ever, there should be protection for this would be the important, the power half, of the lateral movement. Sicher's theory seems to have made no provision for nor given any thought to protection against "strain in the capsule" when the patient turns on the power in mastication:

(1) Does the working condyle maintain its lateral and forward position of nonstrain during the power stroke?

(2) If so, what combination of holding muscles keep it there?

(3) How the condyle could hold a lateral position is especially intriguing.

(4) If the condyle does not maintain this nonstraining position during the medial stroke, just when does it find its way back to the position of "strain" from which it must again be removed before the jaw may safely begin the next radial lateral?

(5) When it does return to this detrimental position, just what purpose is served?

Directional Forces of Jaw Movements

In plotting the resultant directional forces of the jaw muscles in combination, it is discovered that the "retracting fibres" of the temporalis find

[13]McCollum, Beverly B.: Fundamentals Involved in Prescribing Restorative Dental Remedies. Reprint from June 1939 to February 1940 D. Items Interest, p. 29.

their nearest opposition in a *fulcrum* supplied by the external pterygoid muscle and the temporomandibular ligament. Both connect at the condyle neck while the temporalis inserts medially and deeply along the coronoid process.

Condyle Drawn Downward—The retractile pull, therefore, of the temporalis has the supplemental effect of drawing the condyle somewhat downward off the fossal slope on an arc limited by the temporomandibular ligament.[14] If there were any undue strain "in the capsule" or on the meniscus, this alone, operating under a reflex, would tend to relieve it.

So-called Retrusive Movement of Mandible—The same downward supplement to the retractile pull of the temporalis provides an explanation for the so-called "retrusive movement" of the mandible about which there is so much discussion and even controversy.

Actual Protrusion Does not Take Place—The mandible does not actually retrude; instead the condyles arc downward slightly as described. This results in a reciprocal tipping upward and fractionally backward at the front of the mandible. In normal dentition the steep inclines of the upper and lower bicuspids are brought into contact while the posterior teeth are left parted slightly. To overcome this and to consummate uniform closure and occlusal contact, it is merely necessary to contract the masseters and internal pterygoids naturally as is done constantly in swallowing or in mastication. Their upward and forward pull repositions the back of the jaw and the condyles and then closes the posterior teeth along a normal closure arc.

Illusion Produced—Since the lower bicuspids are meanwhile sliding up the forward-slanting inclines of their maxillary opponents the *illusion* is produced that the jaw has been in retrusion.

Proof of Illusion—That retrusion of the mandible is an illusion has been demonstrated on witnesses both hostile and receptive until no reasonable doubt remains. McIver[15] has written a paper that includes this observation. With proper respect for the authorities quoted, their various concepts of the Bennett movement can be challenged by at least three arguments.

Opposing Concepts of Bennett Movement—The first argument tending to refute the explanations quoted for the cause of the Bennett movement arises from the fact that it is the nonworking condyle that is being credited with forcing the working condyle and the entire jaw to move radially or medially. But, once more, the nonworking condyle is a universal joint and no universal joint can *force* movement on anything.

Universal Joint is Adaptive Only—Every universal joint is completely submissive to any force imposed upon or sent to it. If one agrees with the generally accepted concept as expressed by Thompson[16] that an abnormal ". . . tooth guidance dominates the normal guidance of the articular eminence, and the condyle is forced away from the articular eminence in function," he can hardly insist at the same time that "The movements of the mandible and the articulation of the teeth . . . are controlled by the temporomandibular joint."[17] *One concept or the other must be wrong.*

Laterals Outside Functional Range—The second argument stems from the now established fact that laterals are not only outside functional range and play no part in function but are also detrimental to articulation. This being so, a concept of the Bennett movement as a component of lateral motions would put the Bennett in the category of useless and detrimental jaw movements.

Bennett a Component of Natural Functional Movements—The third argument comes from the concept that a true Bennett is not a component of laterals at all but of natural functional movements, the major one of which is the vertical movement. In substantiation of this assertion Figure 7 shows two gothic arch tracings.[18] The lower or rearward tracing is the one usually taken at the vertical dimension decided upon for the case. The upper or forward tracing is the one made in accordance with the Stansbery method of securing check bites at cusp height level to complement those secured at occlusion level.

Rotational Centers of Condyles Shift to Left—When the jaw was opened 1½ millimeters to make the upper or forward tracing, the rotational centers of both condyles shifted to the left with the left condyle moving slightly forward while the right condyle moved slightly backward. These conclusions are not drawn merely from the obvious shifting of the tracing apex. That *might* have resulted from misalinement of the tracing plate or it *might* indicate simple deviation from the midline.

Bodily Shift of the Mandible—Proof that the mandible has shifted bodily while opening (the Bennett movement) is demonstrated by the uniform lack of concentricity between the wings. This shows that the condyles carrying the centers have both shifted and that it was not a simple "medial" or "side-to-side" movement; it involved at least two dimensions and possibly a third.

Opening Bennett Reverses in Closing—Obviously, the opening Bennett must reverse itself in closing if the teeth are to interdentate smoothly. Early experiments[19] proving this to be so have been substantiated by five years of practical application and observation on several hundred cases.

Explanation of Cause of Bennett Movement—The cause of the Bennett movement in natural functional motion is not far to seek nor difficult to grasp. It is common knowledge that similar parts of the body (two hip joints, for instance) are never symmetrical. Applied to the jaw joints, it is easy to understand that the condyles and their respective fossae are asymmetrical in shape, size, and head

[14] Page, Harry L.: The Hinge Bow, DENTAL DIGEST **58**:536 (December) 1952.

[15] McIver, L. W.: Analysis of Jaw Relationships in Treated Class II Malocclusion, Angle Orthodontist, in press.

[16] Thompson, John R.: Concepts Regarding Function of the Stomatognathic System, JADA **48**:636 (June) 1954.

[17] Granger, Ernest R.: Functional Relations of the Stomatognathic System, JADA **48**:639 (June) 1954.

[18] Buchman, Jack M.: Relining Full Upper and Lower Dentures, J. Pros. Dent. **2**:707 (Sept.) 1952.

[19] Page, Harry L.: The Bennett Movement, DENTAL DIGEST **57**:413 (September) 1951.

position. There should be no difficulty in visualizing what must happen when a single unit such as the jaw tries to open or close on two asymmetrical bearings.

Inequalities Compensated—If the jaw is to function at all, it must compensate for the unequal radii, shapes, positions, and sizes of the joints by a bodily shift at the fulcral points. The condyles, having their individual fulcral bearing points in different median planes and being loosely encapsulated, can do this readily. Assertions that operation of the Bennett during vertical motion would cause the jaw bone to bend, or that a single unit such as the jaw cannot rotate on two axes do not merit discussion. Absence of a knowledge of head and jaw physiology or of ordinary mechanics is evident.

Conclusion

This article has frankly challenged several popular articulation concepts without offering any substitute or remedy. These have been and will continue to be the subjects of other papers. Regardless of its form or outcome, the hoped-for result is discussion. Nothing promotes knowledge faster than vigorous debate. Articulation progress has been stagnated long enough because of unquestioning acceptance of principles that do not work.

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Factors Influencing Calcium and Phosphorus Metabolism

Hormones

The Parathyroids—In parathyroid insufficiency or under experimental conditions in which the parathyroid glands are removed from an animal, the plasma phosphorus rises whereas the calcium level falls. The output of calcium and phosphorus in the urine, but not in the feces, is markedly decreased. A greatly increased appetite for calcium develops. If treatment is not instituted tetany may result. Administration of an extract of the parathyroid gland, of massive doses of vitamin D, of A. T. 10, or of a high-calcium, low-phosphorus diet seem to be equally effective in maintaining animals in a fairly normal state of health.

Effects of Increase: On the other hand, when the body's supply of the parathyroid hormone is increased either by injection of the hormone or because of clinical conditions of hyperparathyroidism, the plasma calcium may increase to as much as 18 milligrams per 100 cubic centimeters whereas the plasma phosphorus is lowered. A concomitant increase in the kidney excretion of both calcium and phosphorus ensues. The skeletal tissues undergo decalcification.

Chief Role of Parathyroid Hormone: These disturbances in calcium and phosphorus metabolism induced by variations in the quantity of parathyroid hormone in the body are consistent with the present theory that the chief role of the parathyroid hormone is to maintain the calcium and phosphorus level of the plasma. The hormone apparently has a solvent action on bone salts. It also probably is concerned in the mobilization of phosphorus from the labile organic phosphates in the soft tissues. Still another effect of the hormone is the facilitation of phosphorus excretion by the kidney.

Enlargement of Glands Induced: Fluctuations in the levels of calcium and phosphorus in the plasma are attended by fluctuations in the quantity of the hormone elaborated by the parathyroid gland. In order to meet the need for varying quantities of the hormone the glands have the capacity for undergoing hypertrophy, the hyperplasia being characterized by an increase in the number of chief cells and by a disappearance of the loose architecture of the gland. Thus a condition of hypocalcemia resulting from a low-calcium dietary or one of hyperphosphatemia, such as found in chronic renal insufficiency, will induce the enlargement of the glands.

Factors in Reduction of Gland Size: The hypertrophy of the glands noted in ricketic rats occurs only in the low-calcium, high-phosphorus type of diet which results in a plasma low in calcium and high in phosphorus. Moreover the hyperplasia noted in fetuses of parathyroidectomized pregnant rats can be prevented by feeding the mother a high-calcium, low-phosphorus diet. On the other hand, a low-phosphorus diet in animals has been accompanied by a reduction in gland size. It is interesting to note that vitamin D therapy will prevent the gland enlargement resulting from a low calcium diet as well as reduce further the hypoplastic glands resulting from a low phosphorus diet.

The Thyroid—In cases of thyrotoxicosis, large quantities of calcium appear to be lost by way of both the intestinal tract and the kidney. However, no one as yet has been able to demonstrate a correlation between the basal metabolic rate and the quantity of calcium or phosphorus thus excreted—a relationship which would seem to be essential, if the increased excretions were provoked by thyroxine, per se. That the effect really may be a secondary one is seen in experiments which demonstrated that thyroxine-treated rats consumed enough extra food to account for the increased fecal losses and enough extra water to account for the increased urinary losses. Some experiments have been reported, however, which suggest that in children calcium utilization and retention may be influenced somewhat by thyroid administration.

The Pituitary—Hypersecretion of the growth hormone of the pituitary gland appears to increase calcium and phosphorus excretion by way of the kidney but not the intestine. In the serum the normal concentration of these two elements is not disturbed. The marked osteoporosis in giants may be the result partly of a calcium intake insufficient to calcify the rapidly forming bones. When the pituitary gland is removed, on the other hand, the level of inorganic phosphorus in

(*Continued on page* 407)

ECTOPIC CALCIFICATION *and* FLUORIDE INTOXICATION

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DIGEST

Fluoride when ingested in natural form, that is, fluoride that has passed through the metabolic processes of fish, plant, or animal, has totally lost its active calcium-combining danger, and is thus quite different from the fluoride supplied artificially which invariably retains some of its calcium-active property and is therefore harmful to ingest. Mottling of the teeth is not an isolated phenomenon. The purpose of this article is to establish the biologic significance of the toxic or calcium-active fluoride fraction, and to prove that this fraction alone is responsible for the toxic effects generally attributed to the total intake.

Biochemical Factors

Experimental evidence has shown[1] that a reaction will occur between calcium phosphate or bone and sodium fluoride in which phosphate is displaced. The evidence further shows that the reaction will go to completion at infinite dilutions of the reacting substances because bone adsorbs calcium fluoride (one of the end products of such a reaction) progressively. Whether this withdrawal is temporary or permanent does not affect the issue.

Effects Vary in Degree—The biologic consequence that is observable when the concentration of the calcium-active fluoride fraction in water is high will be present only to a lesser degree if the concentration is infinitely low.

Intoxication Cumulative and Progressive—It has been established that fluoride intoxication is both cumulative and progressive.

Mottling Unreliable as Clinical Guide—External mottling of teeth has been adopted as a clinical yardstick to measure or make the distinction between what is admitted to be a toxic dose and a dose which is supposed to be nontoxic. But it has been shown[6] that mottling of enamel is by no means a reliable criterion and that ectopic calcification of the pulp tissue is a more reliable guide because it is applicable to teeth which are already fully erupted, and indicates the type of progressive tissue change that is to be expected from fluoride intoxication.

Clinical Signs of Disease—In previous writings attention has been drawn to the occurrence of ectopic calcific pulp changes occurring in the teeth of children and adults living in fluoride-in-water areas. The importance of these changes cannot be overemphasized. They are a clinical sign of disease.

Physiologic and Pathologic Changes—For clear understanding of the following discussion it is necessary to draw a distinction between calcification and ossification, and to establish the difference between what may be described as secondary (physiologic) and the ectopic (pathologic) changes that are due to fluoride intoxication.

Distinction Made—Wells, quoted by Bertwhistle,[3] makes the following clear distinction:

(a) In calcification lime salts always remain in clumps and masses, often fusing to greater or lesser extent, but never with profuse even penetration as in ossification.

(b) All the cells in the calcified area, if not dead, disappear for the most part, and there remains a perfectly inert mass, virtually a foreign body, instead of a specialized tissue as in ossification.

(c) Ossification is accomplished only in varieties of connective tissue, but calcification may involve any cell or tissue provided it is sufficiently degenerated.

Ossification usually follows calcification, but this is not invariably the case.

Preliminary Degenerative Change—In secondary dentine formation (Fig. 1a) the irritant or toxin usually arises and enters from without, and the preliminary calcification is usually followed by ossification.

Possible Source of Degenerative Change — Preliminary degenerative change may arise from normal aging. Whatever the cause, however, it can be clearly distinguished from ectopic calcification by the fact that (a) secondary ossification arises from, and is invariably connected with, the original structure, and (b) although it may show irregularities and atypical form, it nevertheless possesses a structure of some kind characteristic of the original pattern.

Sign of Interference with Normal Tissue Function—In ectopic calcification (Fig. 1b) such as the calcific plugs or "islands" about to be illus-

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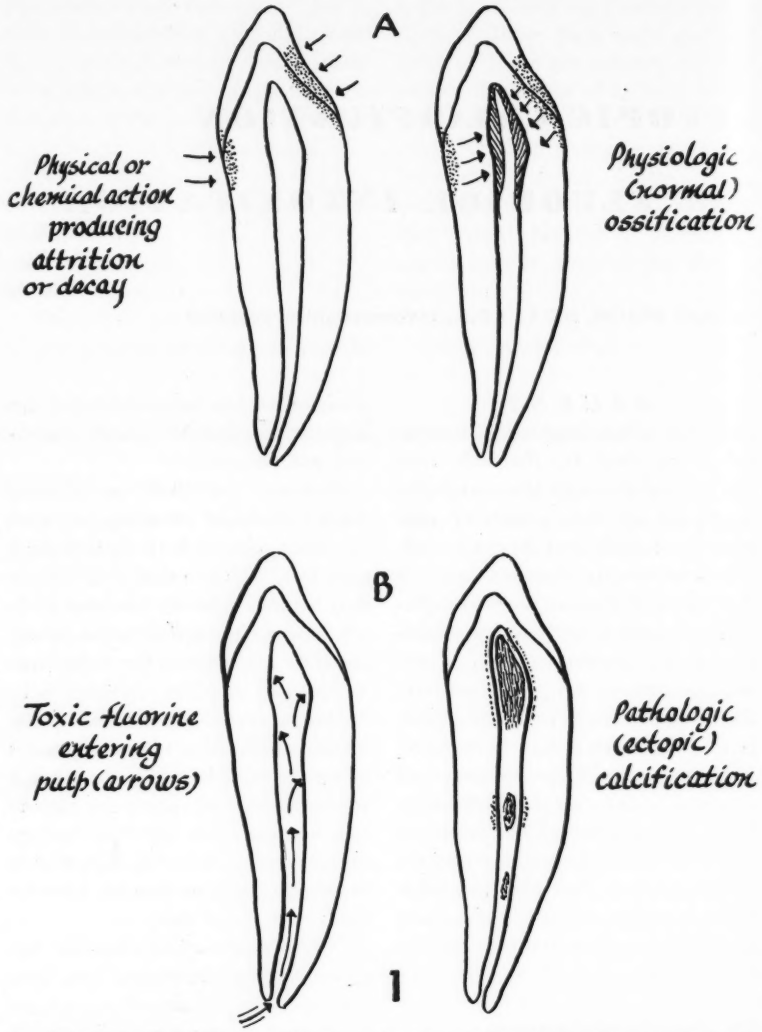
trated, and which are characteristic of fluoride intoxication, the toxic elements are brought to the pulp chamber via the blood vessels: (a) the resultant calcific degeneration has no connection whatsoever with the original dentine, and (b) it is entirely structureless and does not ossify. This degeneration is a pathologic sign of interference with normal tissue function.

Effects of Metabolic Process in Relation to Dose—The advocates of fluoridation have not attempted to draw a distinction between the fluoride that has passed through the metabolic processes of fish, plant, or animal, and fluoride which is in its calcium-active form. They can therefore have no true conception of what is to be regarded as a toxic dose.

Stored Fluoride Regarded as Foreign Body—It has been proved[1,6] that only a part of the fluoride which is absorbed into the blood stream is excreted and that teeth and bone retain calcium fluoride in proportion to the total intake of fluorides from *every* source. Although these facts dispose of McClure's claim that fluorides ingested below a certain level are totally excreted, the fact that bone and teeth retain calcium fluoride is no longer considered of basic importance. The fluoride which is stored is to be regarded only as a foreign body, although it may be an additive factor if it is stored in excess to the damage arising from calcium-active fluoride.

Fate of Living Tissue—In other words, the fluoride that is excreted and the fluoride that is stored bears a relationship only to the total intake from *all sources*. The fate of the living tissue depends solely on the type of fluoride salt which reaches the blood stream.

Detoxification at Expense of Tissue Elements—A calcium-active fluoride salt which reaches the blood stream will displace a proportionate amount of phosphates from the system and in the chemical process will be converted to calcium fluoride and thus become detoxified at the expense of the tissue elements of the body. The resultant calcium fluoride has no further property to react with tissue.



1. ***Illustrates the difference between the physiologic (normal) secondary dentine formation and the pathologic ectopic calcification arising from fluorine intoxication. At (a) secondary dentine arises from and is directly connected with the original dentine. As the condition progresses, the pulp chamber gradually narrows to a slit opposite the area of decay or attrition. In (b) the calcified areas have no connection with the original dentine, are usually multiple (except when fused) and are found along the course of the blood vessels.***

Safeguarded by Natural Processes—The fluoride that is stored in the bones and tissues of one animal has no toxic effect on another animal when it is consumed in the form of food. It is scientifically immature to introduce a chemical which is known to be toxic to the vital processes of the human body.

Clinical Factors

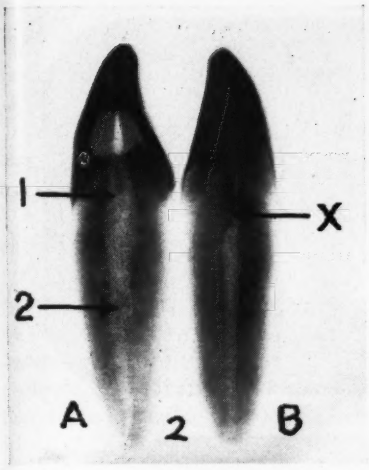
The following observations may be regarded as axiomatic:

"Cell degeneration is of prime importance to the pathologist and to the clinician, for it constitutes disease."[4] (Figs. 1-6).

"There is no isolated phenomena in tissues. The death of an element reacts upon the functioning of its neighbors and upon the character of the surrounding medium. This common law must be applied to bone as to all tissue."[5] (Figs. 1-6).

The Ionic Hypothesis and the Toxic Fraction

It is universally stressed that sodium fluoride added in minute quantity to a public water supply loses its

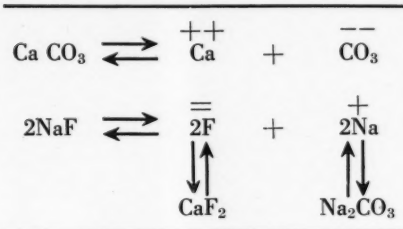


2. ***Illustrates a clinical example of the diagrammatic presentation in Figure 1. (a) A tooth taken from a man resident in West Hartlepool (England) where the water supply contains a total of 2.6 ppm fluorine, including a calcium-active fraction of 0.39 ppm fluorine. The tooth shows multiple ectopic plugging of the pulp chamber completely isolated from the original dentine. Note also in this and other illustrations that far from narrowing of the pulp chamber, there is actually a dilation and absorption of the original dentine opposite the calcific areas. This fact is of considerable academic interest. (b) Shows the tooth of a man of approximately the same age with no fluoride in his water supply. Note the secondary dentine formation opposite the area of abrasion and attrition resulting in a narrowing of the pulp chamber to a mere slit. The secondary ossification is a continuation of the original dentine. (Reproduced by courtesy of*** THE DENTAL PRACTITIONER)

identity. This misleading statement has been widely used by the advocates of fluoridation of public water supplies.

Reaction with Calcium Salts—Depending on the concentration of sodium fluoride when it is added to the water supply and the manner in which it is added, the sodium fluoride may lose some of its identity by reaction with the calcium salts in the water supply.

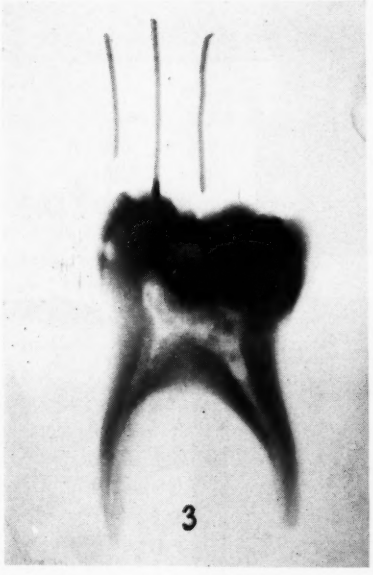
Maintenance of Identity in Equilibrium—(1) When no more calcium fluoride falls out of solution in the form of a precipitate, or (2) when the concentration of sodium fluoride is sufficiently low, then the remaining sodium fluoride will retain its identity in a state of equilibrium as shown in the following equation.



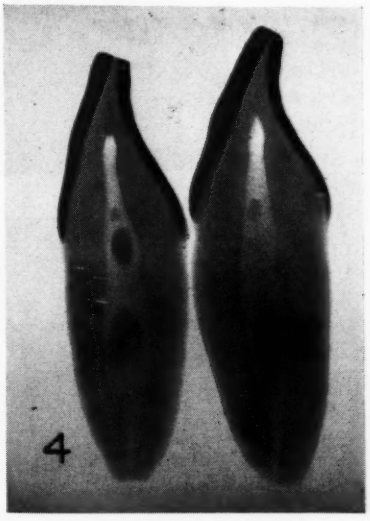
Any concentration below 13 ppm

Ionization Tendency Equals Recombination Tendency—From this statement alone it is obvious that the argument that sodium fluoride loses its identity in water cannot be sustained. How can loss of identity occur when the tendency of a salt to ionize, at whatever level of concentration, is as strong as its tendency to recombine? The ionic hypothesis states that sodium fluoride in ionizing becomes resolved into sodium ion (+) Na and fluoride ion (—) F, nothing more or less than this. Ions are not to be confused with atomic sodium Na and molecular fluorine F_2.

Equilibrium Unaffected — When, therefore, a state of equilibrium has been reached, we have the various substances in solution with their respective pairs of ions. It matters not how much calcium salt is present in the water, this state of equilibrium will remain unaffected as long as the respective ions retain their charge or unless one of the products of the equation, for example calcium fluoride, is withdrawn from the sphere of influence.



3. ***This is an x-ray print of a deciduous tooth from a boy of eight years living in an area where the water supply contains 0.82 ppm fluorine, including a calcium-active fraction of 0.33 ppm fluorine. The multiple calcific deposits are identical with those exhibited by a youthful industrial worker in an aluminum factory (Fig. 4). This suggests that the water supply in South Shields, England, has a toxic effect on the inhabitants identical to that caused by industrial fluoride. Compare this figure with the illustrations in "The Pathological Significance of Mottled Teeth".[6]***



4. ***An x-ray print of a young industrial (aluminum) worker, aged 23, showing multiple calcific plugs identical with those previously shown in teeth from fluoride-in-water areas, and with the tooth in Figure 3. There is no trace of enamel mottling.[6] Her home water supply contains 0.18 ppm fluoride. This suggests that a fraction of the fluoride present in the "natural" water supply of South Shields and other endemic districts is identical in chemical nature with that derived from the aluminum industry and has the same biologic effect.***

5A, 5B, 5C, 5D, 5E, *and* **5F** *The patient whose lower incisors are illustrated in Figure 4 is suffering from generalized degenerative changes of the periodontal membrane resulting in multiple pocketing. Space will only permit comment on the x-ray of the lower incisors (Fig. 5D) although all the teeth show ectopic plugging. The patient has been working in an aluminum factory for four years. Note ostitis of bone and eroded appearance of the alveolus. There has been no history of a blow or trauma to these teeth; the pocketing on the lingual side reached the apex of the teeth which had to be extracted because of pain and tenderness. Although it appears that there is sufficient alveolus to support the teeth in a firm condition, yet in common with other similar cases, the pocketing, although discreet, was deep and penetrating resulting in the loss of two teeth. Plugging of the pulp chamber can be seen as dense areas obliterating the pulp tissues, but as shown in this figure and in Figure 4, the calcific deposits are lying as "islands" in the pulp chamber unconnected with the original dentine.*

Salts may be Absorbed into the Bloodstream—If a water supply containing a calcium-active fluorine salt in such a state of equilibrium is drunk, the human body may absorb any or all of the salts shown in the equation into the blood system.

Misconception—The body does not absorb ions haphazardly, like drawing from a lottery. If the body absorbed ions haphazardly, the effect of administering any given salt would be unpredictable. If the body absorbs sodium fluoride into the blood stream, that sodium fluoride will *then* react with body calcium at all levels of concentration however minute, for it has been shown experimentally that bone *absorbs* calcium fluoride and retains it as a solid progressively. Because this is equivalent to precipitation, the reaction will go to completion.

Calcium and Phosphorus Balance Upset—In this way the sodium fluoride will finally lose its identity by being *wholly* converted into calcium fluoride within the body with damage to calcium and phosphorus metabolism. Dentists are reminded that it is only necessary to reduce the level of ionized calcium from 4.25 milligrams per 100 milliliters to a value below 4 milligrams per 100 milliliters, to arrive at a value which is considered low and associated with ill health.[7]

Toxic Fraction may be Separated—It has been proved experimentally that it is possible to separate that fraction of the total fluorine content of the water supply which is calcium active. An explanation of this has been presented previously.[2,6]

Problem of Mottled Teeth—The dental and medical professions are now confronted with the fact that in some "natural" water supplies capable of mottling teeth or bringing about ectopic calcification in the pulp of teeth, a chemically active fluorine salt of the same chemical nature as that liberated in artificially fluoridated water, or in industrial processes, is present.

6. ***Teeth from a patient of 42 years from the South Shields area. All the teeth are apparently sound. The patient suffered from advanced periodontal disease. Note that opposite the calcific plugs the pulp chambers are dilated because of an absorption of the original dentine to accommodate the calcific deposits. (Reproduced by courtesy of*** **THE DENTAL PRACTITIONER**)

General Conclusions

Evidence has been presented to show that the biologic effects produced by industrial fluoride are identical with those produced by the calcium-active fraction of fluoride in drinking water which is capable (1) of mottling teeth, (2) of producing ectopic calcification in the pulp of teeth, or (3) of bringing about a degeneration of the periodontal fibres surrounding them.

On the basis of these facts it is estimated that a far smaller concentration of fluoride than has been thought previously to be harmful is responsible for mottling of teeth in endemic areas.

It is hoped therefore that the tradition that no drug will be administered to anyone, *the dose of which can neither be determined nor controlled*, will continue to be honored.

Fort William, Caladh, Scotland

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VITAMIN A

and Its Effect on Oral Structures

JACK A. WEICHMAN, D.D.S., Tujunga, California

DIGEST

This article describes the specific changes in tissue, teeth, gingival and subgingival tissue from a lack of vitamin A in the diet. The effects of replacement therapy in these classifications are indicated.

Physiology of Vitamin A

Vitamins are involved with the biochemical mechanisms concerned with the conversion of food and energy into the many forms of activities essential to normally functioning organisms. The pathologic consequences of Vitamin A deficiency are similar in man, monkey, cattle, swine, dogs, rabbits, guinea pigs, rats, mice, and fowl. For these and other reasons facts about Vitamin A revealed in animal experiments are without doubt applicable to man.

Vitamin A and Carotenoids

Vitamin A is a derivative of carotenoid pigments from plants. As far as is known, vertebrates cannot synthesize carotenoids *de novo*. Probably all higher animals can convert certain carotenoids to vitamin A.

Wide Distribution in Plant World—Carotenoid pigments, yellow and red compounds, are widely distributed in the plant world from bacteria and diatoms to garden fruits and vegetables.

Provitamins—Out of this large group four substances are known at present from which animals can form vitamin A. These have been named alpha, beta, and gamma carotene and kryptoxanthin, and they are called provitamins. A preparation of carotenoid, echineone, from the sex glands of the sea urchin has also been found active as a provitamin A. The term carotene is used here to represent this group of provitamins.

Storage of Vitamin A In the Body

Approximately 95 per cent of the total quantity of vitamin A in the body is present in the liver, with small amounts in the lungs and kidneys. Most animals have a remarkable capacity for the storage of vitamin A; a rat may in a few days store enough vitamin A to supply its nutritional needs for several months. In times of low intake this store is used to supply physiologic needs. During a prolonged deficiency the reserve is gradually depleted, normal cell functions are suspended and pathologic changes develop. In animals as well as in human beings, the vitamin A content is much lower in the liver at birth, irrespective of the diet of the mother, than in the liver of the normal adult. Accumulation of vitamin A in the liver tends to increase with age and is dependent on the character of the diet.

Tissue Effects of Vitamin A Deficiency

The seat of physiologic disturbances in vitamin A deficiency is in the epithelial cells. Chemical roles are suppressed but proliferative powers are not inhibited; nor are the potentialities of cells lost, as is shown by the retina, to normal physiologic functions when vitamin A is restored.

Epithelial Tissue—The primary effect of vitamin A deficiency is on epithelial structure. The sequences are atrophy of the epithelium concerned and the substitution for it of a stratified keratinizing epithelium, identical in appearance in all locations and arising from focal proliferation of basal cells.

Atrophy Appears: The absence of vitamin A causes starvation specific for many epitheliums and this in time leads to atrophy and to a state wherein the cells, although having the appearance of viability, become inert in physiologic activity and in their role of covering membranes. An invariable sequence in pathology is that a break in the continuity of a tissue is followed by reparative proliferation. In vitamin A deficiency the basal cells normally concerned in maintaining the integrity of epithelium respond by mitotic division.

Areas of Proliferation: The effect of the deficiency after atrophy is the appearance of scattered areas of proliferative activity beneath the original epithelium. The new (reparative) cells by their continued growth undermine and replace the original epithelium and, regardless of the previous function and morphology of the region, develop into a stratified keratinizing epithelium. Both in experimental animals and in infants an atrophic epithelium having the histologic appearance of viability may remain for a period above the replacement epithelium.

Continuous Process: The replacement epithelium is identical in all locations and comparable in all layers with epidermis. It has a continuous layer of basal cells (a stratum germinativim) in continuous proliferative activity while superficially it is con-

tinuously casting off keratinized cells.

Gross Pathologic Conditions: The striking gross pathologic conditions of vitamin A deficiency in animals and in man are the outcome of the accumulation of keratinized epithelial cells in glands and their ducts and in other organs. In glandular organs, cysts of considerable size are formed, filled with yellowish cheesy masses of keratinized cells. In the lungs of human beings as well as in experimental animals this process leads to cyst formation, bronchial occlusion, and consequences such as bronchiectasis and atelectasis.

Distribution of Keratinizing Metaplasia—The effect of vitamin A deficiency on epithelial structure is called keratinizing metaplasia. In human infants, keratinizing metaplasia has been found in the conjunctiva, mucosa of the nares, accessory sinuses, trachea, bronchi, pancreas, renal pelvis, ureter, salivary glands, uterus, and periurethral glands.

Early Signs: The commonest and earliest appearance of metaplasia is in the trachea and bronchi. Next in frequency and sequence is the pelvis of the kidney. The early effect of the deficiency on the respiratory mucosa is an explanation offered for the frequency, severity, and persistence of the pneumonias that have been in most instances responsible for death in vitamin A deficient infants.

Involvement of the Eye: In the human being involvement of the eye is late. The first change is metaplasia of the epithelium of the cornea and of the conjunctival sac. Atrophy and metaplasia of the ducts of the paroocular glands contribute to the consequences of the accumulation of keratinizing cells in the conjunctival sac. The cornea becomes vascularized, edematous, and infiltrated with leukocytes. Infection of the cornea favored by the excessive accumulation of keratinized cells may lead to ulceration of the cornea and hypopyon.

Order of Response in the Rat:

(1) Salivary glands, including submaxillary, parotid, and all accessory glands of the tongue, buccal cavity, and pharynx.

(2) Respiratory tract, including nares, maxillary sinuses, Jacobson's organs, trachea, and bronchi.

(3) Genitourinary tract, including the renal pelvis, bladder, ureter, epididymis, prostate, seminal vesicles, coagulating glands, uterus, oviducts and accessory sex glands of the vulva.

(4) Eye and paroocular glands, including the corneal and palpebral conjunctival and the Harderian, intraorbital and extraorbital lacrimal glands and the meibomian glands.

Deficiency Effects on Teeth—Owing to atrophy and metaplasia of the enamel-forming organ, the continuously growing incisors of rats and guinea pigs are profoundly affected. After the atrophy of the enamel-forming organ there are atrophy and cessation or depression of the functioning of odontoblasts. The formation of enamel stops and striking deformities of the tooth take place because of diminished and defective formation of dentine.

Atrophy of Enamel: The enamel organ which extends for the entire length of the incisor tooth on its labial surface early undergoes atrophy and metaplasia and consists finally of atrophic remnants of the epithelial papillae and squamous cells replacing the ameloblasts and stratum intermacium.

Enamel-Forming Organ Renewed: The anterior part of the enamel organ is first affected but finally the whole length is involved, including the basal formative end. At the base end there is a continuous renewal of the enamel-forming organ and in many rats with marked metaplasia in many organs virtually normal structures were found here, though with distorted relationships owing to pressure deformity of the tooth because of defective or absent dentin formation.

Enamel Organ Affected by Metaplasia: Metaplasia of the ameloblasts affects the enamel organ, and odontoblastic activity is organized by the presence of ameloblast cells. Thus if the ameloblasts are abnormal they may be unable to control the odontoblasts, with a resulting abnormality in the odontoblasts also. The odontoblasts show marked reduction in size and loss of their normal epithelial arrangement. The atrophy of the odontoblasts, with the exception of those opposite the enamel organ, proceeds rapidly and is complete usually when general histologic changes in many organs are well established.

Alteration of Odontoblast: During the atrophy the odontoblasts continue to form an irregular, poorly calcified dentin matrix. All cells resembling odontoblasts disappear, and the internal surface of the dentin becomes bounded by cells that are indistinguishable from the other connective tissue cells of the pulp. The odontoblasts also showed more severe alterations on the lingual side where the dentin was thin, folded or absent, than on the labial side where the dentin was excessively wide.

Odontoblasts Incorporated into Dentin: During the process of atrophy, odontoblasts, and rarely capillaries that supply them, become incorporated into the dentin because some cells remain functional for a longer time than others and by continuing to form, have surrounded nonfunctioning cells with dentin. In advanced deficiency there may be gaps in the wall of the dentin which always occur near the basal or open end of the tooth.

Vascular Inclusions in Vitamin A Deficiencies: These are characteristic and differ from those seen following parathyroidectomy. In the latter condition, the vascular inclusions recur at more or less regular intervals and penetrate the dentin in an almost straight line. The vascular inclusions in vitamin A deficiency are more numerous and branch within the matrix of the dentin. In addition, especially deep vascular inclusions occur consistently at the cementoenamel junction and represent a most severe alteration in the dentin formation.

Displacement of Pulp Cavity: A characteristic displacement of the pulp cavity over to the lingual side is found in vitamin A deficiency. An invasion of epithelium which arose from the lingual odontogenic epithelium occurred on the pulp. A continuously proliferating epithelium appears to survive quite readily in the nutritive connective tissues of the pulp. The

epithelium here does not become a stratified squamous type, and it also shows no tendency to keratinization or cyst formation. There is a characteristic tubular arrangement, however, which is suggestive of a glandular organ and which reminds one of the epithelial cords and rests observed normally in the peridental membrane.

Epithelium Found in Adjacent Tissue: Some of the proliferating epithelium, however, still possesses the chemotactic properties of stimulating dentin formation. This capacity is weak and defective so that the result is not a true dentin product, but an irregular, amorphous, and unorganized matrix. The amount of pulpal epithelium increases with the duration of the survival period. In chronic vitamin A deficiency extending over a long period, the epithelium is found in the adjacent tissue.

Osteoid Tissue also Found in Pulp: In this situation the occlusal end of the pulp chamber was being constantly filled in by bony material (osteodentin). The sequence of the pulp ossification is the following:

(1) Minute spherical globules of hyaline matrix appear between the pulp cells and simultaneously narrow zones of similar material form about occasional capillaries.

(2) These deposits increase and incorporate pulpal cells which assume the morphology of osteoblasts, and finally, cells in contact with the periphery acquire the morphology of osteoblasts and presumably function as such.

Lack of Color in Incisor Teeth: The absence in places of the thin outer layer of the enamel which is composed of parallel prisms in which yellow pigment occurs may account for the lack of color in the incisor teeth of A deficient rats. After long periods of an A deficient, postweaning diet, the incisor teeth frequently become completely white. The calcification of teeth in vitamin A deficient rats is also noted as being abnormal. This is due not only to the abnormality of the cells which produce the matrix, but also to the interference with the ameloblasts.

Degeneration of Enamel Organ Affects Bone: The alveolar bone overlying the enamel organ appears to be adversely affected by the degeneration of the latter in extreme cases. The cementum becomes notably wider as does the labial alveolar periosteum. The outline of the periodontal membrane is extremely irregular and is generally narrower than normal.

Gingival and Subgingival Tissues—In vitamin A deficient animals varying degrees of epithelial hyperplasia and cellular infiltration of the corium (the connective tissue between the gingival and subgingival tissue and the alveolar bone) were observed together with an increased susceptibility to periodontal disease. The subgingival epithelium in deficient animals becomes hyperplastic either in the form of a single localized or general increase in the number of epithelial cell layers, or as finger-like projections into the subjacent connective tissue of the corium and downgrowths along the cementum. In the cementum there is frequently an associated increase in its cellular components, especially in the immediate vicinity of the hyperplastic epithelium.

Effects of Vitamin Deficiency on Soft Tissue: These are not easy to distinguish because of the influence of external physiologic and possibly chemical and bacterial factors, but it would seem that hyperplasia, including thickening of the keratinous surface layer, is the usual sequel.

Effect of Replacement Therapy

The lesions of vitamin A deficiency, uncomplicated by destruction of tissues by infectious processes, disappear rapidly after restoration of vitamin A to the diet. In rats (1) gain in weight, (2) resumption of growth in bones and teeth, and (3) regeneration of the bone marrow and spleen become apparent in from six to eight days.

1. Epithelial Tissues: Reparative changes in the metaplastic epithelium begin as early as the fifth day. The initial changes are separation of superficial keratinized cells and vacuolization of cells of intermediate layers. The epithelium becomes divided into two zones by further vacuolar degeneration and leukocytic infiltration. The superficial zone of cells degenerates; the deep zone of cells corresponding to the stratum germinativum of the epidermis, survives and the cells proceed to differentiate into the type of epithelium originally present.

2. Teeth: Restoration of the enamel organ takes place first at the basal formative end. The odontoblasts in juxtaposition to the enamel organ at this end recover and soon assume normal morphology and function, as shown by the deposition of predentin and canaliculi. The predentin calcifies rapidly and secondary dentin or osteodentin grows in size because of the deposition of dentin upon them. The columnar odontoblasts reappear, first at the basal formative end and then work forward. The connective tissue cells in contact with the dentin become plumper.

Comment: The presence of normal odontoblasts in replacement therapy does not necessarily represent a recovery of atrophied odontoblasts. They rather appear to be new and young odontoblasts that have become differentiated from the mesenchymal cells under the influence of the now normally functioning odontogenic epithelium.

3. Gingival and Subgingival Tissues: Repair of gingival and subgingival tissues occurs simultaneously with recovery of teeth from a vitamin A deficient diet. Characteristics of the gingival and subgingival tissues of animals receiving an abundant supply of vitamin A are the following:

(a) A thin keratinous outer layer of the gingival epithelium.

(b) Thin and well-formed gingival and subgingival epithelium with little or no prolongation into the underlying connective tissue or along the cementum.

(c) No cell infiltration into the corium.

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Factors Influencing Calcium and Phosphorus Metabolism

(*Continued from page* 398)

the plasma is reduced and the phosphatase level elevated. In addition, a negative calcium balance has been observed, but not a negative phosphorus balance. This negative calcium balance can be restored to normal by the administration of the growth hormone, which will also correct the decalcification of teeth induced by the removal of the gland.

The Ovary—Estrogenic hormones in clinical doses have little effect on the plasma calcium and phosphorus in man. When administered as replacement therapy to women with postmenopausal osteoporosis, however, the estrogenic hormone has been reported to induce retentions of both minerals. But in normal young girls following the menarche, estrogen therapy has been observed to reduce the percentage retention of calcium. Calcium retentions, moreover, appear to be lower during the first half of the menstrual cycle than during the two weeks following ovulation. This suggests again an antagonistic action of the estrogen toward calcium utilization.

Dietary Factors

Lactose—Lactose in contrast to the other carbohydrates commonly eaten by man, increases calcium utilization in children and growing rats and dogs and in parathyroidectomized dogs. Although its effect is not so great as that of vitamin D, that effect is exerted even in the presence of vitamin D. The feeding of lactose to preschool children receiving adequate vitamin D has resulted in a 33 per cent increase in the amount of calcium retained. This effect on calcium metabolism has long been ascribed to the establishment in the intestinal tract of an acidophilic type of bacteria; these are said to produce sufficient acid to change the hydrogen ion concentration of the tract and so increase the absorption of calcium. This explanation, however, does not accord with recent data.

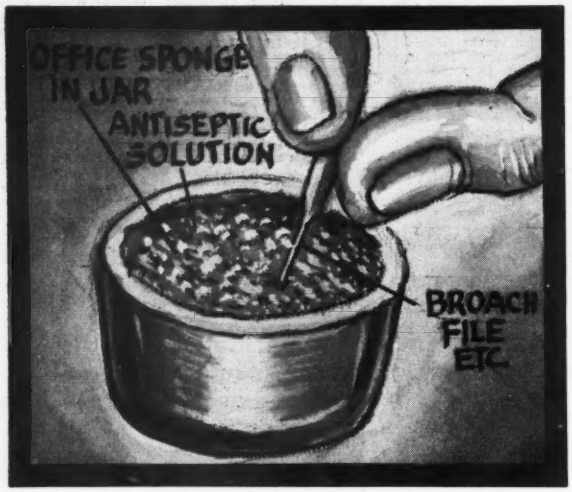
Calcium Loss—In preschool children mentioned above, the greater retentions of calcium were paralleled by a decrease in the amount of calcium lost by way of the kidney. Moreover, the feeding of lactose or its hydrolytic products has not been found to change the extent of absorption of calcium from the intestinal tract of rats. These findings suggest that lactose exerts its effect by making more utilizable that calcium which has found its way into the blood stream.

Protein—A beneficial effect has been reported regarding protein on absorption and retention of both calcium and phosphorus by adult human subjects. But no study has yet been reported in which the protein moiety of the diet was the only variable; the beneficial effect observed may have been due to some factor other than protein per se in protein-rich foods.

Fat—Fat appears to have an influence on calcium metabolism. Investigators observed that best absorption of calcium occurred when the calcium intake bore a definite relation to the fat intake, the most favorable results being obtained when babies consumed 130 to 170 milligrams of calcium and 4 grams of fat per kilogram of body weight and when older children on a mixed diet consumed 85 to 140 milligrams of calcium and 13 grams of fat per kilogram. The amount of calcium lost in the form of soaps by the babies studied in this investigation did not exceed 30 per cent of the intake. Pure stearic acid, in contrast to fats, has been reported to be excreted largely as the soap. In celiac disease and in idiopathic steatorrhea, negative calcium and phosphorus balances have been found, a result probably due to the formation of the insoluble calcium soaps.

Wheat—Whole wheat apparently contains a substance which has a deleterious effect on calcium utilization. In a study with rats, better bone calcification was found in a group of rats fed white bread containing milk solids, both breads having an identical calcium content. Moreover, in a study of ten adult human subjects, observers found calcium absorption markedly reduced when white bread, contributing 40 per cent of the total calories, was replaced by whole-wheat bread. The calcium requirement of these ten subjects appeared to approximate 600 milligrams daily when white bread was eaten; it averaged 1 gram daily when brown bread was eaten. Although the calcium in whole-grain cereals exists partly as the calcium phytate, this is probably not the explanation for the poor utilization of the calcium.

Adapted from "Dietotherapy" by Michael G. Wohl, Philadelphia, W. B. Saunders Company, 1945, pp. 180-182.



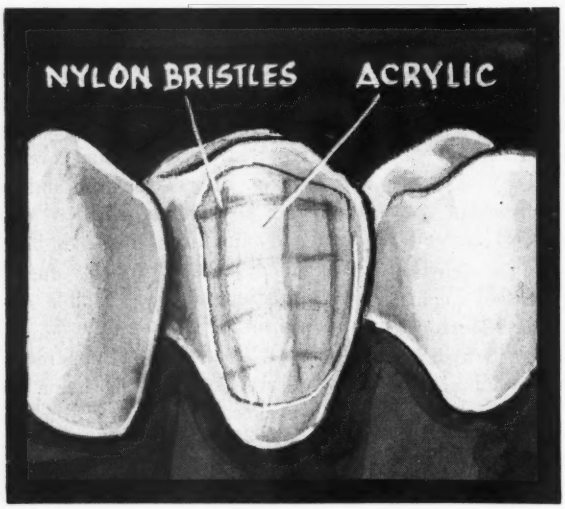
1

Clinical and Laborator

Cleaning Root Canal Instruments

Sidney D. Fleiss, D.D.S., Richmond Hill, New York

1. A stamp-wetting sponge in a glass container is saturated with an antiseptic solution. After use in the root canal, plunge the broach or file into the sponge. This will remove the debris that is adherent to the instrument.

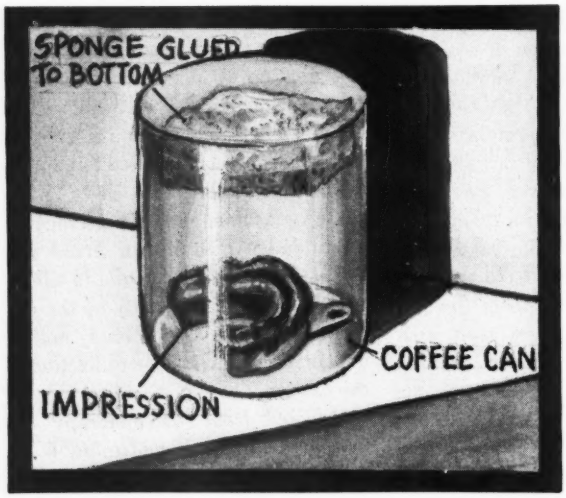


2

A Wax Pattern for a Veneer Crown

Richard M. Adams, D.M.D., Chatham, Massachusetts

2. Bristles from an old nylon toothbrush inserted into the wax pattern are suitable to form struts and spurs for the retention of acrylic in the gold casting.



3

Preventing Drying of Hydrocolloid Impressions

L. C. Holtzendorff, D.D.S., Valdosta, Georgia

3. A sponge is glued into the bottom of a coffee can. The sponge is moistened and the can is inverted over the hydrocolloid impression that is on a flat surface.

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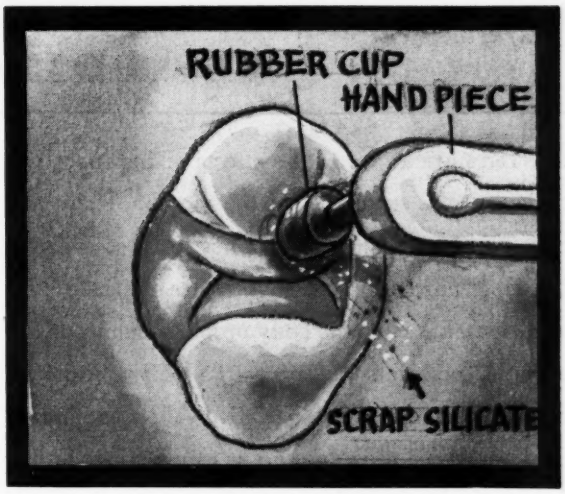
High Lustre for Amalgams

Sam R. Adkisson, B.S., D.D.S., Conway, Arkansas

4. After the amalgam is polished with pumice a high lustre may be obtained by applying scrap silicate cement powder on a rubber cup.

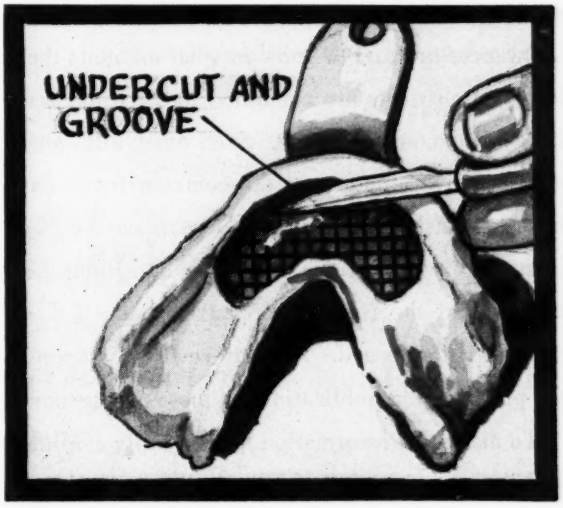


4

Impressions for Partial Dentures

M. E. Whiteside, D.D.S., Storm Lake, Iowa

5. Make a snap impression in alginate. With a sharp knife remove the material where the teeth are present and some material over the ridge area. Scar the surface of the impression to improve retention. Make another mix of alginate and place it in the preliminary impression. This two-stage impression assures more accurate results.

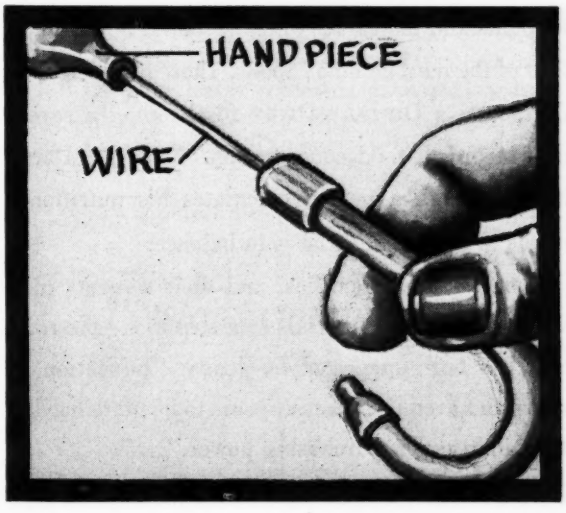


5

To Clear a Clogged Saliva Ejector

Harry W. Tustison, D.D.S., Dania, Florida

6. When unable to push a cleaning wire through the clogged saliva ejector, place the wire in the straight handpiece.



6

technique involved; and jot down the advantages of the technique. This shouldn't take ten minutes of your time. Turn to page 414 for a convenient form to use.

Send your ideas to Clinical and Laboratory Suggestions Editor, DENTAL DIGEST, 708 Church Street, Evanston, Illinois.

The EDITOR'S *Page*

DENTISTS are aware of the effects of improper nutrition on the structures of the mouth. There are both positive and negative values to be considered in proper nurtriture. Positive values emphasize the need for taking into the body proper amounts of the essential nutrients to satisfy the daily minimum metabolic requirements. Negative values emphasize the importance of restricting or eliminating certain foodstuffs that are injurious to body cells.

To follow a regimen of proper nutrition it is necessary to know what the recommended daily dietary allowances are and to know in what amounts these essential nutrients are contained in the usual servings of the common foods. Such daily allowances and nutritive amounts in the common foods have been established by the U.S. Department of Agriculture Bureau of Human Nutrition and Home Economics and the National Research Council Food and Nutrition Board. This information is open to the public in the publications of these two agencies.

To make this information more readily available and in convenient form, the editorial staff of DENTAL DIGEST has created a DIAL OF NUTRITION based on official publications. This DIAL gives the caloric amounts and the values of ten essential nutrients in 200 of the most common foods. There has also been developed a DIETARY DIARY for keeping a seven-day record of food consumption. From the DIARY and the DIAL a person calculates his nutritional status to determine if he is in balance.

Dentists, their families, and their patients may wish to use these materials as a step along the road that has been suggested by Jeans: "Education in nutritional requirements appears to be more needed than increase in purchasing power."

Nutritional imbalances are common at all levels of the population and a careful study made among 610 men who are physically fit for daily work in industry shows just how common nutritional injury may be observed. These are the generalizations reached in this study:[1]

"1. No cases of acute nutritional deficiency diseases were observed.

"2. About one-fourth of the men gave some evidence of suboptimal nutrition with respect to one or more nutrients, particularly with respect to calcium, phosphorus, thiamine, riboflavin, and ascorbic acid . . . Suboptimal ratings were found for one or more vitamins in 26 per cent (158 subjects) of the sample. Of these 158 subjects, one had a suboptimal rating for vitamin A, ten for thiamine, 58 for riboflavin, 45 for niacin, and 84 for ascorbic acid.

"3. Suboptimal nutrition, also obesity, were observed widely throughout all education, age, income, and ethnic groups.

"4. Dietary faults frequently observed were low consumption of milk and vitamin C rich vegetables and fruits, a high consumption of unenriched bread and pastries, sweetened beverages, and candy bars.

"5. Inadequate breakfasts and between-meal snacks of sweet foods were contributing causes of unbalanced diets."

The experience of every dentist will confirm the results of this study. At least 25 per cent of our patients are suffering from nutritional injury and suboptimal nutrition. We have a magnificent opportunity to be leaders in health education on this vital subject, for: what is good for the teeth is good for the rest of the body and what is good for the organism-as-a-whole is good for the tissues of the mouth. The human body is a totality.

[1]Babcock, N. J., et al.: Nutritional Status of Industrial Workers, Milbank Memorial Fund Quarterly **32**, No. 4 (Oct.) 1954.

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MEDICINE and the Biologic Sciences

The Problem of Pernicious Anemia

Seldom is pernicious anemia seen in persons under 30. It is much more common in those over 50. The average age at time of diagnosis is 55.

Formerly, the life expectancy of a person with this condition was short, often a few months or years at best. With the discovery of the beneficial effects of whole liver and the subsequent development of liver extracts as substitutes for liver, this picture has changed. Now, the life expectancy often exceeds that of other persons of the age group.

Pernicious anemia is a familial disease. It is often present in two or more siblings and in two or more generations of a family. Usually it occurs as a result of a congenital defect in the gastric secretory mechanism with achlorhydria as the most readily demonstrable feature. The most frequent evidence of the disease is anemia resulting from the inability of the bone marrow to produce cells due to lack of material necessary to form erythrocytes. The mucous surfaces of the mouth, particularly of the tongue, may be irritated and sore. This may be a sign of a deficiency in vitamin B complex. The central nervous system may be so affected as to produce neurologic symptoms, the most disturbing of which is difficulty of locomotion, rarely with complete loss of locomotor ability.

A satisfactory therapeutic substance must control, to an optimal degree, all manifestations of the disease except the congenital gastric defect. Whole liver does this quite well. Extracts of liver are as effective as whole liver and greatly simplify treatment for patients.

Folic acid held great promise as a substitute. It was found, however, that folic acid does not maintain normal blood levels in all patients nor does it control neural manifestations. There is evidence available to suggest that it is possible not only to control anemia with vitamin B_{12}, but also to prevent development and progression of neural manifestations. Further critical study is necessary before vitamin B_{12} is accepted as a complete substitute for either whole liver or liver extracts.

Hydrochloric acid in doses of 20 drops or more, well-diluted with water and taken with meals, will often control the persistent diarrhea present in some patients. Intercurrent illnesses may be treated as in any other patient without fear of disturbing effectiveness of the liver substance. The quantity of antipernicious anemia substance should be increased rather than decreased during treatment of acute infections and during the course of surgery. A balanced diet of muscle meat, vegetables, fruit, milk, and eggs is just as important today as it was in the days of whole liver therapy. Limitation of excessive amounts of fat and concentrated carbohydrates is desirable.

Neither life expectancy nor economic or social activities of the patient should be limited by pernicious anemia, except in the rare instance in which severe neural disturbance is present before intensive treatment is started or when uncontrollable complications appear. Most patients live long and useful lives.

Murphy, William P.: Pernicious Anemia as a Problem in Geriatrics, Geriatrics **9**:99-105 (*March*) 1954.

Drooling Associated with Cerebral Palsy

Drooling is the act of allowing saliva to drip out of the mouth. Normally, the saliva is pushed to the back of the mouth by the posterior tongue muscles accompanied by closing of the mouth and contraction of the pharyngeal muscles in a normal swallowing movement.

The condition is noted in most children with cerebral palsy. Formerly it was regarded as a sign of mental deficiency. Often it is the cause for social rejection. Actually, it is merely a symptom of some physical deficiencies in the muscle control of the mouth, jaw, and throat.

Children with cerebral palsy who drool are characterized by (1) poor mouth control, (2) poor tongue control, and (3) other factors concerned in a normal swallowing pattern. Usually these children are mouth-breathers, but this is not necessarily because of enlarged tonsils and adenoids, though such is frequently the case.

The drooling is stopped by mastery of the normal swallowing pattern plus learning to breath through the nose with the mouth closed. One method of teaching correct swallowing is the application of a jaw mask that blocks the entrance of air through the mouth but permits breathing through the nose.

The mask is made of a piece of horsehide lined with softer leather to fit around the chin and over the mouth to include the upper lip. The nostrils are left free for breathing. A piece of sponge rubber is placed inside and underneath the leather lining to block the air and secretions from the mouth opening more effectively. Pieces of old inner tube are used for elastic straps to hold the mask in place. The rubber straps are placed over the crown of the head to give a slight pull to mouth closure. The saliva, which then collects in the mouth, can still run forward. It is sealed off by the sponge rubber pad in the inner side of the mask and can not escape from the mouth. When enough saliva has collected the child

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is literally forced to swallow and empty the mouth with three or four gulps. After a while he learns to swallow regularly to avoid this collection at the front of the mouth.

The mask is worn for as long as 30 minutes at various times during the day. It can be worn usually while the child is doing other activities such as walking, training, or hand skill. It is estimated that a child swallows at least 120 times every one-half hour. There is a good carry-over after the mask is removed. Most children gradually develop the habit of swallowing their saliva instead of drooling.

When a mask is removed after a treatment period, the inside is wet where the saliva has been blocked. As the child improves there is less and less dampness on the inside of the mask. When the child succeeds in keeping his mask dry, the treatment is stopped as drooling has been mastered by the child's own reflex efforts.

Dorinson, S. Malvern: Antidrool Mask for Children with Cerebral Palsy, JAMA **155**:439-440 (*May* 29) 1954.

Tuberculosis in the Aged

One of the most serious problems in tuberculosis control is tuberculosis among the aged. There are many aged persons who are carriers of tubercle bacilli. These are dangerous contacts, especially for children.

Today over 50 per cent of all tuberculosis deaths occur after the age of 45. It is believed that the greater prevalence of tuberculosis now reported among the elderly is due to (1) refined diagnostic procedures, (2) chest roentgenogram surveys, and (3) to the fact that more people attain old age. The longer persons infected with tubercle bacilli live, the more time there is for clinical tuberculosis to develop in their bodies. The age period between 55 and 65 is important in that it contains the largest number of aged tuberculous persons. The mortality rate among elderly men patients is much higher than that among women patients.

Contrary to popular belief, resistance to tuberculosis is decreased rather than increased in old age. The immunity acquired in earlier years does play a role in the chronic course of the disease. Patients who develop adult tuberculosis after the fifth decade usually do poorly. The pathology of pulmonary tuberculosis in the aged differs in no detail from that of tuberculosis in younger persons. The pathology of old age, however, has a marked effect on the pathology of tuberculosis and is especially responsible for the high mortality.

The resistance of the aged is less effective than in earlier adult life. In tuberculosis, an elevation of the number of blood lymphocytes is associated with resistance to the progress of infection. There is evidence that elderly patients require more ascorbic acid to keep the plasma level normal, a factor in tuberculosis resistance.

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(*See pages* 408 *and* 409)

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culosis mortality and morbidity rate is to be realized, we must be alert to the fact that tuberculosis not only exists in elderly people but is definitely increasing. If large numbers of our elderly tuberculous patients are allowed to remain undiagnosed and untreated, no control program can be effective.

Andosca, John B.: Tuberculosis in the Aged, Geriatrics **9**:332-333 (*July*) 1954.

Psychologic Reaction to Disease

It is important that the physician understands the emotional problems of his patients so that undesirable behavior trends can be avoided or contained before barriers to favorable progress develop. The mechanistic concepts of disease should be moderated with an understanding of psychosomatic principles. Time and personal reserve must be sacrificed to supply the needs of the patient.

Some emotional manifestations are directly stimulated by organic somatic difficulty. Disturbed emotional states and behavior patterns may be caused by (1) protracted pain, (2) metabolic and hormonal upheavals, or (3) other physiologic derangements. Fever and toxic states impart feelings of unreality and may disturb defensive and inhibiting mechanisms. Such manifestations are unavoidable and may be controlled only by appropriate and effective therapy of the somatic state.

Most untoward reactions are psychogenic in origin. These emotional tensions must be curbed since the psychologic manifestations may (1) aggravate somatic disorders and cause further patho-physiologic disturbances, and (2) prevent the patient from observing the therapeutic regimen necessary for treatment. In many chronic diseases, these mechanisms eventually lead to complete invalidism.

The mental image the patient has of the body is a basic factor in all

illnesses. The concept of body image involves such factors as (1) health, (2) strength, (3) cleanliness, and (4) efficiency. Preservation of the total body image in the highest state of esthetic beauty and vital functioning is of great concern to all. Any illness or operation threatening the continued integrity of the body scheme may cause emotional problems.

The effect of the threat to body image depends upon the preexisting personality structure. Persons who were previously emotionally unstable become completely disorganized, while those with healthy defenses to utilize may be scarcely affected.

Terror and disorganization accompany acute illnesses. The patient fears death or chronic disablement and has not had the opportunity to get business and spiritual affairs in order.

When a chronic illness exists, a new person is created out of the old psychosomatic assets and disabilities with an entirely different set of relationships toward self and other people. Old conflicts and emotional patterns are used to crystallize attitudes to meet changed circumstances.

Intense anxiety can be tolerated for only a certain length of time. Eventually the patient will employ some technique to relieve the stress. A basic mechanism for such release is the obliteration of inhibiting mechanisms and the relinquishment of adult behavior.

Good patient relationship demands honest understanding and good communication. Patients endure anything better than uncertainty.

Boyd, David A.: Psychologic Needs of the Medically and Surgically Ill Patient, Wisconsin M. J. **53**: 158-160, 169-175 (*April*) 1954.

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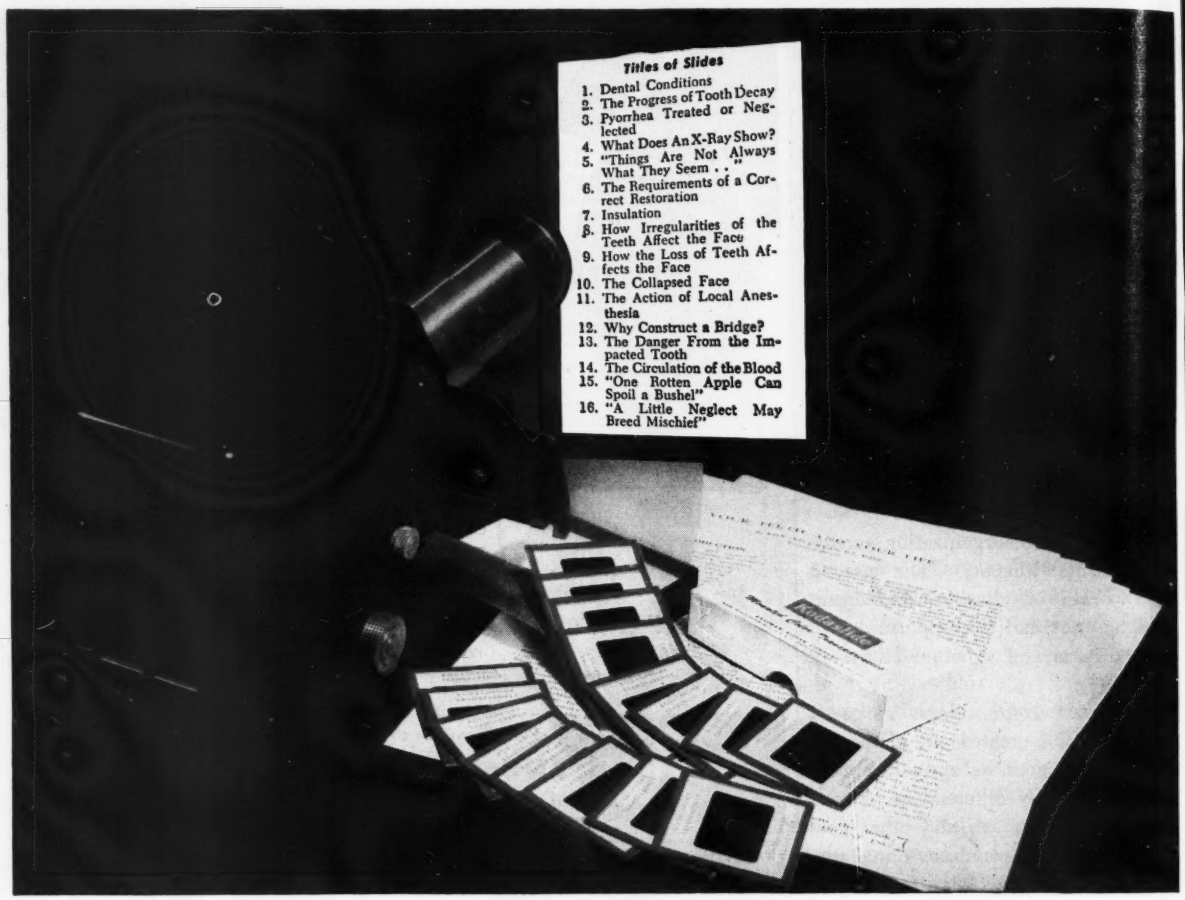
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The Voice in Diagnosis

After practicing dentistry for thirty years I am still at a loss to explain the success of some dentists and the abject failure of others. I have known excellent operators and clean-cut men who labored a professional lifetime at the ragged edge of bankruptcy. Other, lesser men with restricted skills waxed prosperous. One dentist comes to mind who was never able to escape from a little office over a store in a run-down neighborhood. This dentist was one of the best I have ever known. Then there is the one who is a composite of all incompetent things but who has a show place for an office and a full galaxy of associates, hygienists, and assistants around him.

The first fellow in the down-at-the-heel neighborhood is a decent chap but he is afraid that somebody will find it out. He scowls and snarls in the aggression that is frequently the mark to cover one's own fears and inadequacies. People have been known to concede that he is a good dentist despite his ulcerous personality.

The other lad with a smile and a gush and a chair-side manner has covered himself with annuities and other paper of fortune, although his classmates wonder how he passed the first course in freshman technique. He is that fumble fingered! I have been around him enough to know that he is not endowed with any technical skills except the one of making money which he has developed to hypertrophic proportions.

What makes some dentists *click* and others *clank?* The ones that click are usually out-going in their personalities, which is to say that they are constantly attentive to the interest of the other fellow. They are attentive to the other person's word,

In your ORAL HYGIENE this month

Correcting Some Misconceptions on OASI

Whether you are for or against the inclusion of dentists in the Old-Age and Survivors Insurance plan, you will want to read the article "Correcting Some Misconceptions on OASI."

Doctor Wayne Clark believes that many dentists are basing their objections to OASI on inaccurate or obsolete data. He says, "I was misled by this information for a long time, but at long last I awakened, went down to my local social security office and got the facts about Old-Age and Survivors Insurance." He quotes and refutes five erroneous statements which have been widely circulated and which may have influenced many of the dentists who are now opposed to OASI.

★ ★ ★

"Are You Conducting a Limited Practice?" Doctor H. Keith Terrill intimates that some dentists curtail their effort as well as their field of service. So-called limitation of practice may, he says, be just a graceful way to avoid doing dental work that requires extra study and skill. "It is a poor service you render to the patient, and a sad commentary on yourself, if you stopped learning when you graduated from school. It takes a little nerve to tackle the difficult or unusual . . . But if you keep on trying and learning, you will be a better dentist and a better man."

★ ★ ★

Stuart Covington warns that "Your Assistant is Listening." Not that you need fear her conscious actions—it is her *un*conscious *re*actions that may lead her to make casual remarks that could be misunderstood or misinterpreted. Safeguard yourself against this innocent source of unfounded rumor.

★ ★ ★

Lankenau Hospital's dental division head, Doctor Conrad F. Hellwege, believes in visual aids to implement the hospital's program of "Health Through Education." A tooth several feet high lights up section by section to show enamel, dentine, pulp cavity, gum tissue, and supporting bone. A gigantic head with a "see-through" oral cavity duplicates the human mouth in all details. There are 70 displays (both medical and dental) in the unique health museum that characterizes this progressive hospital in suburban Philadelphia. Charles P. Fitz-Patrick makes the report.

★ ★ ★

Do you know "The 'How-To' of Good Patient Relationships for Dentists"? Doctor Charles L. Lapp discusses eight rules for achieving a pleasing chair-side manner. The eighth is especially important: "You must look at yourself as a professional salesman, and not just as one skilled in operative proficiency."

★ ★ ★

"Wilbur", the dentist who always knows the right answers, is talking about "wills" this month. If you haven't been following this humorous presentation of serious management subjects, you will surely want to meet Wilbur in this issue. Doctor Robert P. Stickley is his creator.

★ ★ ★

Other articles and all of the regular monthly features are waiting to give you information, help, and reading enjoyment, in the September issue of Oral Hygiene.

respectful of his ideas and opinions, quick to grasp and make capital of any weakness in the other's ego. "Panderers" they are called by their detractors. It isn't fair, however, to attach such an unsavory label to a person who has developed the knack of getting along with the other fellow. Some people are so naturally extroverted that they are acting naturally when their dour opposites think they are putting on an act. The social personality is not a dishonest or insincere one. It is just different from the ingrown one.

The extrovert is skillful in the interpersonal relationships because he likes the impact of other people's ideas and feelings. As a dentist he may be sympathetic and understanding which are two of the virtues high on the list of qualifications compiled by patients. Sympathy means the skill of *feeling with another*. It means the ability to feel with the patient who is faced with pain, misfortune, or unpleasantness. Sharing feelings are among our most pleasant moments. When we share a great and pleasurable emotion with another person we are near our highest level as a human being. Even higher perhaps in the scale of civilized being is the ability to feel the heartaches, the sorrows, the frustrations of another person. It is easy to share pleasure; so hard to share pain.

The dentist who has developed the skill of sharing the feelings of fear, anxiety, or apprehension as they are presented by human beings coming to him for treatment has a quality that will help the dentist along the road to success. People will often prefer him to one more technically proficient.

The anxieties of people who come for dental treatment are shown in their gait, their posture, their face, their voice. The fearful person cringes in the attempt to make himself less conspicuous to blows and to reduce the area of his body surface that may be exposed to attack. The anxious person slinks into the dental chair where he sits in contracted posture or grips the arm of the chair in white-knuckled tension. His face is pale, his eyes are dilated, perspiration moistens his brow. With the rapid pulse and increased respiration rate these are all the signs of the dominance of the sympathetic side of the autonomic nervous system.

In another way the anxious person reveals his feelings: by his voice. An otolaryngologist has written a fine book that should concern every dentist who is interested to know the role that the voice plays in human behavior. ("The Voice of Neurosis," by Paul J. Moses, M.D., New York, Grune & Stratton, 1954.)

Dentists are working, not only with the patient who conveys his feelings through his voice, but the dentist is working with some of the physical units that *produce* the voice. A knowledge of the structures of the voice and its function should concern us.

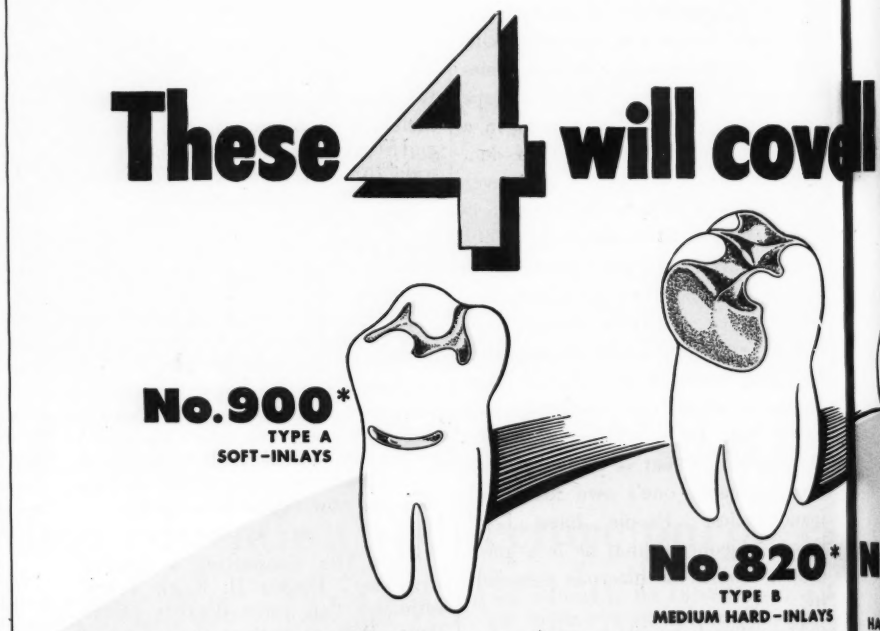
Doctor Moses shows that the voice:

a) Tells something

b) Reveals something about the speaker

c) Elicits reaction from the listener

The "telling something" is the verbal content. The "revealing and eli-



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citing" are the vocal components. The word content (the *matter* of the speech) is often very far less significant than the vocal factor (the *manner* of the speech).

People as dental patients often *say* one thing—a pleasantry of greeting or a comment on a current event, for example—but their *meaning* as conveyed in their voice may show tension, hostility, or anxiety. The clinician should learn to listen to the voice as much as to the words, for of the two the voice may be the more reliable index of the emotion that is being expressed. Moses says: "The importance of divorcing the voice from the context of the words and the difficulty of so doing cannot be stressed enough."

Another quote from this fine book by Moses will give an idea of the wisdom and clear psychologic orientation that the author uses to approach the clinical evaluation of the human voice:

"When an orator approaches the end of his speech, the audience anticipates it through the key and pitch of his words more than through content: this is somewhat similar to the return to C major in music, which can indicate the festive final chords. It means finality, accomplished fact, in speech as well as in music. How diffferent is this end in the neurotic! Indecision, fear, anxiety, guilt do not permit the 'C major' which is combined with a definite lowering of the last syllable meaning the end of a declarative sentence. A statement is accepted as such only if the last syllable has this 'true ring of conviction'; analytically speaking, the lowest tone in the sentence with increased chest register. Under anxiety, the patient does not commit himself to this finality. Figuratively he keeps the door open not by lowering the final syllables, and immediately the statement assumes the melody of a question: rising melody at the end of a sentence, in the last syllable of a word, or in the last vowel in monosyllables. The width of the interval indicates the emotional meaning, the curiosity.

"It is not only the pitch of one syllable that may fall and rise but so may also the melody of an entire word, clause, or statement. A declining melody, particularly if it is repeated regularly, connotes depression. A classic example of this is the famous speech of Edward VIII in which he abdicated for the 'Woman I love.'

"Perhaps the greatest variability is found in its intervals—the difference between pitches of individual syllables. Wide intervals characterize excitement, including excitement from toxic influences such as alcohol and drugs. All the manic emotions are channeled through comparatively large intervals. In operatic music one hears a parallel evidence: in recitativo the small intervals represent facts, thought, remarks. In arias the large intervals express feelings.

"The ambivalence of melodious expression must guide the analyst in his evaluation. Small intervals can mean modesty and orderly realism. But the voice as well may be utilitarian, puritanistic, secretive and cold. A voice that uses large intervals

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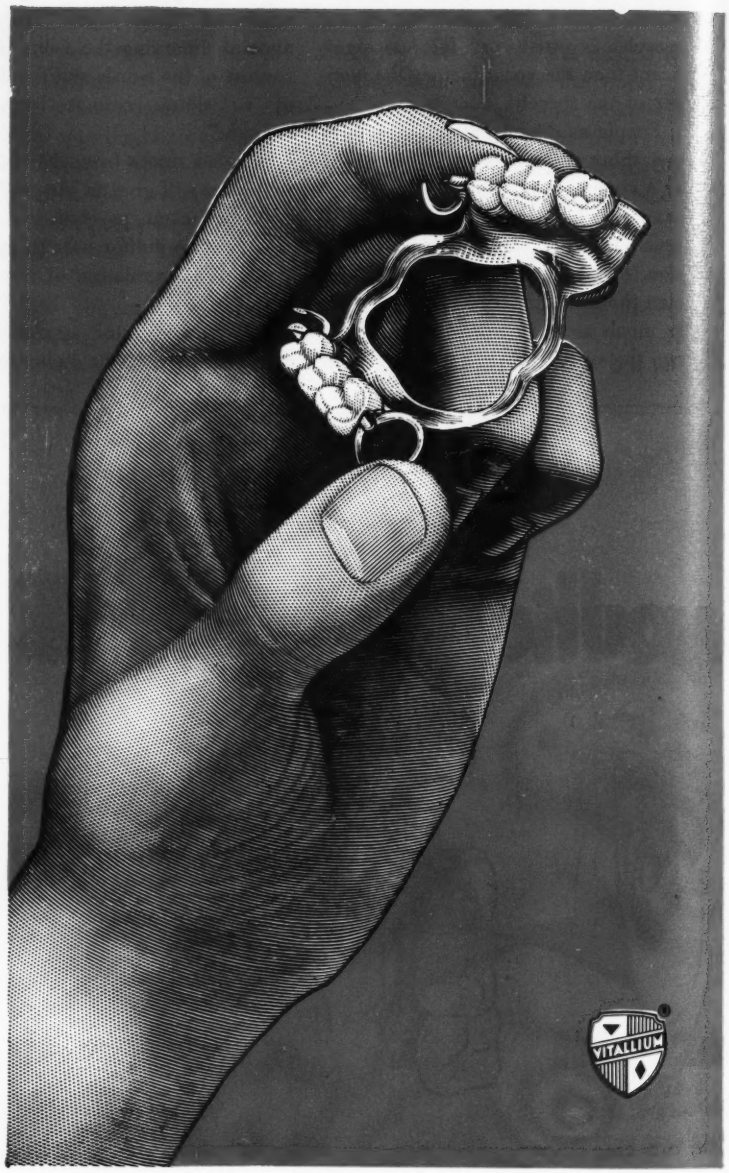
freely and frequently is expressive of emotions, genuine or simulated, which in other words means that the person is rich in feelings or that he has the power of representation or both. On the negative side, large intervals may mean pomposity, exaggeration, or lack of emotional balance.

"If the melody of depression is constant in a voice, the person reveals a pathological symptom. Adaptability to situations will be expressed through a flexible voice. Stiffness suggests resistance to influence, stubbornness or reserve."

We have learned to look with a magnifying lens at the hard and soft tissues of the mouth. We interpret the meaning of the shadows in x-rays. Bacteriologic and biochemical tests have been developed that will aid us in diagnosing. But let us never forget to *look at* and to *listen to* the person who is the patient.

The totality that is the organism, the entity, has been impressed upon us as important at arriving at a diagnosis. We have become mindful of the psychosomatic components in disease. The voice affords us another important clue in diagnosis. The patient reveals feelings and values in his voice. We must remember that the relationship is reciprocal: the dentist also reveals his feelings, his attitudes and values to the patient. While we are making a diagnosis the patient is also making an evaluation. The voice is a two-way diagnostic circuit.

—E. J. R.



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2. Teeth that are continually sensitive to thermal changes are the result of the violation of many details.

3. If the improper cementing media is used, pulp death may occur. Germicidal cements have proved to be the most satisfactory in past years.

4. Make every effort to provide proper temporary protection for the preparation during crown construction. If this is not done, permanent injury to the dentinal tubules may occur.

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crowns, as trauma is an all-important factor in pulp deaths.

7. During tooth preparation, raise the temperature of the applied water spray as high as the patient can tolerate it comfortably. Keep this temperature constant until the preparation is complete. It is my belief that this method is to be preferred over intermittent cooling. Such a procedure has an expanding and contracting action on the pulp.

From J. Pros. Dent. 4:99 (*January*) 1954.

Vitamin C and Infections

VITAMIN C in addition to influencing wound healing is believed by many to combat infection. The cause of this possible effect is obscure; but ascorbic acid (vitamin C) in the presence of oxidising agents degrades many polysaccharides. Polysaccharides in bacterial-cell walls might therefore be attacked; and the bactericidal effect of a mixture of ascorbic acid and oxidising agent might be due to this action. A further possible effect is on mucin. Despite its useful lubricating functions in the body, mucin may aid infective processes by protecting pathogenic microorganisms from the attack of the body defenses and by enhancing their virulence. Therefore ascorbic acid together with oxidising agents, by attacking local accumulations of mucin, may promote resistance to infection and recovery from attacks by fungi, trypanosomes, bacteria, and viruses.

Treatment with ascorbic acid and percarbonate has been reported to result in rapid improvement in stomatitis, gingivitis, and oral mycosis; and it has been suggested that topical application to epithelium-covered surfaces may be helpful in many infections. But, since mucoids in the tissues might be attacked, it would seem advisable to exercise caution in applying this treatment to mucous membranes.

Adapted from *Lancet* No. **6861**: 443 (Feb. 26) 1955.

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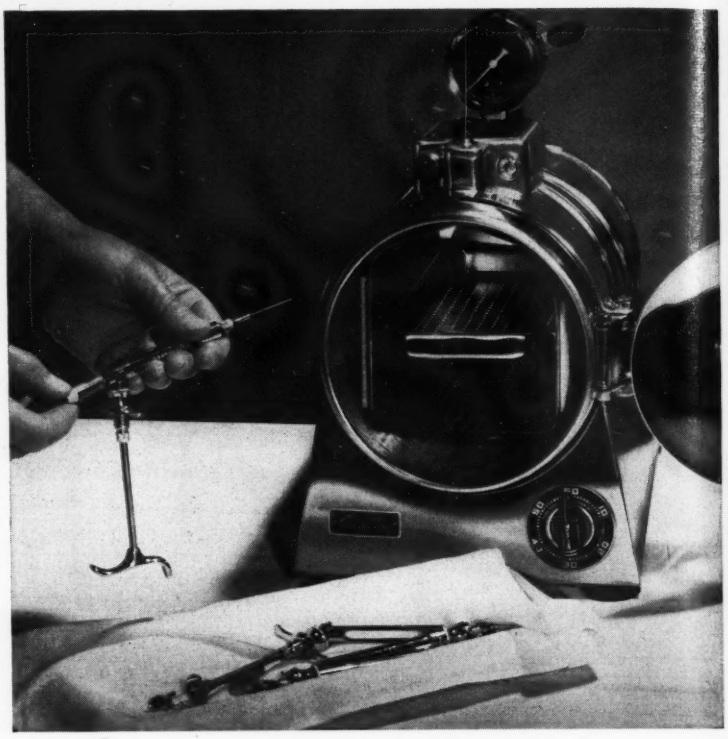

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